Idiopathic Ulcerative Colitis

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IDIOPATHIC ULCERATIVE COLITIS

(DYSENTERY)

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PREFACE

A MORE than usually severe outbreak of what is variously termed ulcerative colitis, or dysentery, having occurred in this asylum in the early part of 1895, and the appearance of a similar, though milder one towards the end of autumn, have induced me to collate and arrange the clinical data of a more than ten years' experience of this most distressing and fatal affection, in the hope that the account may prove of some interest.

In private practice it is not very common, the cases met with are sporadic, not of a very severe type, and generally yield to ordinary remedies. The reverse obtains in those institutions in which it is found. I may be pardoned, therefore, for entering, in some detail, into a description of this ulcerative affection of the colon, more especially as in some asylums it in measure appears to be indigenous, and is liable, at different times and under varying circumstances, to outbreaks of great severity, which might even be called epidemic. I am firmly convinced that this affection of the large bowel occurring in communities of the insane, and characterised by intractable purging and very great mortality, is a specific affection, identical in its

origin, symptoms, results, etc., with former outbreaks of dysentery in this country, occurring in prisons, workhouses, and in times of famine, which have been voluminously written upon, and even been the subject of Commissions of Inquiry. For descriptive and clinical reasons, at least, I shall use the terms dysentery and ulcerative colitis as synonymous. This view, however, judging from the death tables of asylums generally, would appear not to be universally accepted, as among the causes of death we find figuring—even in the same asylum — dysentery, colitis, dysenteric diarrhœa, enteritis. This indicates a very loose nomenclature, or else pronounced opinions as to the meaning of these terms. Enteritis may be dismissed at once, as strictly speaking it is an entirely different affection from that of the colon.

I should feel inclined to eliminate the word dysenteric as applied to diarrhœa, or include all diarrhœas to which the word dysenteric may be applied under the heading of dysentery alone. Clinically, perhaps, it may serve a useful purpose, but the difficulty is to decide the demarcating line between dysenteric diarrhœa and dysentery, more especially as we know the latter often commences with what is apparently ordinary diarrhœa. Moreover, in the nomenclature of diseases issued by the Royal College of Physicians, we do not find the word about which exception is taken used as qualifying diarrhœa, and until this is determined and a definite meaning attached to its employment, the term were better avoided, at least in

statistical tables and classifications. We are thus reduced to a choice of two terms, and so long as we clearly understand what is meant by them, no confusion need arise.

If synonymous, then one only should appear in the death tables, but where the two figure, we must perforce conclude that two distinct diseases are indicated. What the distinction between the two is I have never been able to discover, nor have I ever received any satisfactory account of one. In the earliest reports, and even in those of recent date, we find dysentery playing in some asylums a considerable part in the causes of death, but now in many asylums we find the word colitis or ulcerative colitis used, and partly or entirely supplanting the term dysentery. As already indicated, where the two terms appear in the death table it may be taken for granted that they represent two distinct diseases.

In this view of it the disappearance in an asylum of dysentery where formerly it was common, and the substitution of another disease so identical in its symptoms and *post-mortem* appearances, etc., and equally fatal in its results, will be difficult of explanation. That some difference is implied is apparent, and if my remarks on the subject would call forth an expression of opinion from any who have had similar acquaintance with the disease or diseases under consideration, it would help greatly to the solution of the question.

I propose to deal with the subject in the following order:—

Chapter I.—Statistical tables and observations thereon.

„ II.—Association with other diseases.

„ III.—Clinical history, symptoms, treatment, illustrative cases, etc.

„ IV.—*Post-mortem* appearances, with tables.

„ V.—Ætiology.

„ VI.—Summary and conclusions.

To my colleague, Dr J. H. Goodliffe, I must express my gratitude for his kindness in preparing and photographing the microscopical sections, and supplying me with the results of his bacteriological investigations. My best thanks also are due to Dr F. W. Eurich and my brother, Dr Burns Gemmel, for their kindness in revising and correcting the proofs, and to Mr Spencer for the care he has bestowed upon the preparation of the plates.

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County Asylum, Lancaster,
1*st February* 1898.

Idiopathic Ulcerative Colitis

CHAPTER I

STATISTICAL TABLES AND OBSERVATIONS THEREON

In approaching the subject of ulcerative colitis as it occurs in asylums, there are one or two points to which I would direct special attention. Firstly :—it seems to be, in great measure at least, limited to the larger institutions, and of these to some more particularly than to others, cases occurring every year and often accounting for an enormous percentage, as much sometimes as 33 per cent., of the total number of deaths. I have calculated the average percentage which it bears in different asylums, directly and indirectly, to the other causes of death for periods ranging up to 14 years, and in some the ratio is so decided as almost to compel one to the belief that there is something endemic and peculiar to these institutions which does not exist in other asylums. Year in year out it occurs; one year the type mild and with few fatalities, another the reverse, with a high death rate. The milk and water supply proved to be free from contamination, the drainage good—or if defects once existed they have long ago been corrected—and the food (here I admit may sometimes be found an element of uncertainty) both

as to quantity and quality receiving as far as possible careful consideration.

What is the cause? Why does it exist in one asylum and not in another; under what conditions does it develop; what, if any, special type of mental case does it attack; does it exhibit a predilection for any one class of patients in this asylum, and if so, why does that class seem to be peculiarly exempt in another asylum where the disease is present? These are questions which naturally present themselves for elucidation; some of them can only be imperfectly answered; and others not at all.

The ætiology of the disease will be dealt with in a separate chapter, but I may say here, that if from future experiments and research the affection can be proved to be due to a specific micro-organism, then the question is settled; but until then, what we have, at least as practical physicians, to consider, are the proximate causes, such as overcrowding, etc., all of which we shall discuss later on.

Its preference for one domicile over that of another may also be in great measure explained by the existence or otherwise of these proximate causes. The susceptibility to an attack, of one class over another, or in other words its choice of victims, does not seem to be determined so much by mental as by physical conditions, *i.e.*, it does not select one mental class in preference to another, but the feeble and debilitated of all types. In 114 deaths in this asylum, 64 occurred in dements, 17 in melancholics and 13 in general paralytics. The relative frequency in other forms of insanity I have not stated in the text; they are included in the table and only from a statistical point of view.

In the chapter dealing with the ætiology the occurrence of the disease in general paralytics will be discussed; suffice it in the meantime to say that this class is not specially liable to be attacked, a view which is held by some.

While on the subject of the class of patients affected by

ulcerative colitis we may also consider the influence, if any, which length of residence in the asylum, age, sex and former attacks play in its production.

Residence in the Asylum.

Calculating as previously, over a considerable period, the results only represent a rough approximation, and so far as the first factor is concerned seem to indicate that a residence in the asylum to a certain extent predisposes. The average for men is 7 years, that for women 4 years.

It is to be understood, however, that there are notable exceptions of patients, resident for even more than 40 years, passing unscathed through many outbreaks and eventually dying from some other disease. Others, again, admitted during an outbreak, are attacked within a few days of admission and before they have had time to acquire the "Asylum Constitution." There are several cases I can recall of its ending fatally in less than three weeks after admission, but here it must be borne in mind the patients were not in a fit physical state to resist any noxious influence, owing to previous prolonged refusal of food, and exhaustion from acute mania or melancholia.

Influence of Age.

As for age, I have found the average to be pretty much the same for both sexes—50 years for men and 51 for women, the maximum and minimum being respectively 70 and 19 for the one, and 79 and 19 for the other. The disease, however, does by no means spare those of more tender years; but as comparatively few at that period of their existence are admitted here, no deduction or inference can be made.

Influence of Sex.

No special predilection for one sex or the other seems to exist, although the numbers are slightly in favour of the female. This, however, may be at least partially accounted for by the fact that the latter usually exceeds the male population in numbers, and, generally speaking, their ages are slightly greater and their general robustness less.

TABLE I.

TABLE showing deaths in this asylum, with mental condition, during a period of 15 years, from ulcerative colitis, including those in which it occurred as a complication of, or was associated with, other diseases. The table also gives the deaths from all causes, and from general paralysis, in separate columns.

YEAR.	Total Deaths from all Causes.	Total Deaths from General Paralysis.	Mental Condition in Deaths from Ulcerative Colitis.										TOTAL.		REMARKS.
			General Paralysis.	Dementia.	Senile Dementia.	Epileptic Dementia.	Melancholia.	Epileptic Idiots and Imbeciles.	Mania.	Mania, Chronic.	Idiot.	Imbeciles.	A.	B.	
1881	105	39	1	5	...	...	...	...	...	...	...	...	5	6	
1882	96	29	...	2	3	1	4	...	...	...	...	1	7	11	
1883	173	58	4	3	...	1	4	...	1	...	...	...	10	13	
1884	186	54	3	3	...	1	1	...	1	...	...	...	5	9	
1885	127	25	1	...	1	...	1	...	...	...	1	...	2	4	
1886	181	30	2	10	...	...	3	...	...	...	...	...	10	15	In the total column the (A.) Sub-division represents the cases in which death was due directly to dysentery.
1887	204	41	...	7	2	1	...	...	...	1	...	4	12	15	
1888	123	37	...	4	...	...	...	...	...	...	...	...	1	4	
1889	132	20	...	...	...	1	...	..	...	...	...	...	1	1	
1890	132	18	1	1	...	...	...	...	...	...	...	1	2	3	Sub division (B.) includes (A.) and deaths due to other causes, but complicated by dysentery.
1891	124	19	...	..	...	...	1	...	...	...	...	...	1	1	
1892	156	23	...	1	1	...	...	2	1	..	...	...	5	5	
1893	156	27	...	...	1	...	...	...	...	...	...	...	1	1	
1894	151	41	...	2	1	...	1	...	...	1	...	...	3	5	
1895	156	37	1	3	6	3	3	. .	2	2	...	1	16	21	
Total,	...	498	13	41	15	8	18	2	5	4	1	7	81	114	

Contrary to what some writers (*e.g.*, Flint) have stated with regard to dysentery, second attacks are by no means uncommon. I have known many instances of patients being attacked several times, the attacks being separated in some cases by long intervals. A feature to be also noted in this connection is that those who have once had it are often the first to feel the influence of a fresh outbreak. As in erysipelas, one attack does not confer immunity, but rather predisposes to another. In only 5, however, out of the large number of *post-mortem* examinations appended were distinct evidences of old cicatrices found, though this of itself does not negative the existence of former attacks, as these may not have been of sufficient intensity to cause necrosis of the entire thickness of the mucous membrane, and further, many of the *post-mortems* revealed such total recent destruction of the mucous lining of the colon as to render it valueless for evidence one way or the other.

Second Attacks.

Regarding the time of year during which it is most prevalent, statistical evidence seems to point to the first, third and last quarters of the year respectively, as exhibiting not only the largest number of cases, but the greatest fatalities. This is the calculation based on a period of 15 years; but taking individual years, the disease does not seem to follow in this respect any very definite rule regarding its occurrence, though, generally speaking, it favours cold, and in winters of exceptional severity the number of cases and the mortality appear to be correspondingly increased. In the first quarter of last year, for example, all the cases were of a very acute type, 6 deaths occurring, one—to which I shall allude farther on—resulting in death within 40 hours.

Time of Year it occurs.

[TABLE II.

TABLE II.

TABLE showing deaths occurring in the different quarters of the year for a period of 15 years. This table includes deaths from other causes, but in which ulcerative colitis occurred as a complication.

MONTH. YEAR.	1881.	1882.	1883.	1884.	1885.	1886.	1887.	1888.	1889.	1890.	1891.	1892.	1893.	1894.	1895.	TOTAL.	QUARTERLY.
January, .	1	3	..	.	1	1	2	1	...	...	...	...	..	...	...	9	
February, .	2	1	...	3	1	...	1	...	...	...	...	...	...	...	4	12	1st quarter, 34
March, . .	1	1	...	...	1	1	4	..	...	1	...	1	...	...	3	13	
April, . .	1	...	...	1	...	2	2	2	...	...	...	1	..	...	2	11	
May, . .	1	...	...	...	...	1	1	...	...	...	...	...	...	...	1	4	2nd ,, 18
June, . .	...	...	...	...	...	1	1	...	...	...	...	1	...	...	...	3	
July, . .	...	...	4	1	1	2	1	...	...	2	..	1	...	...	2	14	
August, .	...	...	4	1	...	...	2	...	...	...	...	...	1	...	2	10	3rd ,, 30
September,	...	2	...	1	...	...	...	...	...	...	...	1	..	1	1	6	
October, .	...	...	2	1	...	3	...	1	1	...	...	...	...	...	...	8	
November,	...	1	1	...	...	3	1	...	...	...	1	...	...	1	2	10	4th ,, 32
December,	...	3	2	1	...	1	...	...	...	...	...	...	...	3	4	14	
Total, . .	6	11	13	9	4	15	15	4	1	3	1	5	1	5	21	114	114

CHAPTER II

ASSOCIATION WITH OTHER DISEASES

WITHOUT entering into a historical *résumé* of the antiquity and geographical distribution of dysentery, about which information can always be obtained in text-books and special works on the subject, and before proceeding to a description of the different types and illustrative cases, their symptoms, progress, prevention, treatment, etc., I should like to mention, as tending to throw considerable light on its causation, some of the diseases with which it has been and is still associated.

Association of Dysentery with other Diseases.

First, as regards its association with typhus. It often accompanies or follows in the wake of this disease—witness the epidemics in Ireland in 1800-1, 1818-19 and 1846-47; and the history of wars and sieges afford further and abundant proof of this. Like typhus, again, it is promoted by bad hygienic surroundings, such as overcrowding with its attendant evils; and it, for the most part, is limited to, and most severely attacks, the poorer class of inhabitants. It may also strictly confine itself to a certain district of the town, and on the continent, in the old cities, there are not a few houses known as "Ruhr-häuser" or Dysentery-houses, in which the disease is endemic. At one time exposure to cold and damp; at another bad or insufficient food; contaminated water supply; overcrowding in associated dormitories, especially of feeble

Typhus Fever.

paupers and lunatics, alone or in combination, are found precursors of, and attendant upon, the outbreak. Where several causes, each capable of promoting and fostering disease, are present, it is very difficult to assign them their respective shares in the production of this or that particular affection. One cause, however, which seems common to typhus and dysentery, is overcrowding. In regard to this and insanitary conditions generally, as found in asylums, there is one feature which I may say is peculiar to these institutions, and more especially to the larger ones. I refer to the massing together of feeble dements, epileptics and general paralytics. The necessities of these types of insanity, and the demands made on our accommodation, often compel this, and so render in measure unavoidable what is clearly recognised as a defect. To a greater or less extent therefore, this insanitary blot exists, and the total disregard for habits of cleanliness exhibited by the insane, more especially by the types just referred to, intensifies the difficulties of dealing with all diseases in any way dependent upon unhealthy environments. The constant soiling with excrement and urine of clothing, bedsteads and floors—especially where the latter, as is often the case, consist of soft, porous wood, rendering it almost impossible to thoroughly cleanse them—will always prove a fertile source of zymotic disease. Virchow, speaking of infectious diseases in the army, says, in discussing the question of the ground as the source of the poison, "It is least of all a question of a poison essentially inherent in the soil, but far more the men themselves who defile the ground, and thence outwards, in all directions, the water and the air, through their excretion, especially urine and ordure." If this can be said of communities of the sane, how much more does it apply to collections of feeble insane occupying close and ill-ventilated wards and dormitories, and adding to the unhealthiness of the atmosphere by their filthy habits. We credit the dried-up, finely-pulverized sputa of phthisis, wafted about in the air, with being a prolific means of tuber-

cular infection. If, now, this ulcerative colitis or dysentery, by whichever name you choose to call it, be proved to depend upon a micro-organism, the facilities afforded to dissemination and infection by the frequent saturation of the floors, etc., with excreta containing the specific cause, are abundantly obvious and fully ensured.

Regarding the association of the bowel affection with influenza, I do not feel called upon to express too decided an opinion, but it is a fact which I have not infrequently observed. The writings of others, moreover, show that the one followed upon or co-existed with the other. In view of each being of the nature of a catarrh and affecting mucous surfaces, a predisposing cause common to both may be granted—some atmospheric condition, perhaps—though in the bowel affection local factors, such as defective sanitation, contaminated water supply, etc., are also usually existent and play a more important part. I have seen influenza precede or develop contemporaneously with a severe outbreak of dysentery, and *vice versa*. **Influenza.**

With the abatement of the respiratory catarrh, we not uncommonly find the pressure on our infirmaries continuing, owing to the admission of cases of dysentery, not by any means confined to those debilitated from influenza. As already stated, both may arise contemporaneously, one patient being attacked by dysentery and another by influenza. This, as suggested, seems to lend to the belief that they may have something in common, or even, under certain circumstances, be identical in origin. May the combination of gastro-intestinal and respiratory signs and symptoms described as pertaining to influenza not cease to exist, and the noxious influence expend its full force and energy on the tract, which at the time may be in such a state of lowered vitality as to render unnecessary its travelling farther afield for more pasturage? This supposition of a common origin, of necessity implies that dysentery may be caused, *per se*, by atmospheric influences—that it is air-borne. Against this view, however,

the serious objections may be urged, that it is more dependent upon and usually traceable to local conditions, and that it does not, like influenza, attack wide areas or districts embracing a scattered population.

Dysentery occurs along with malarial disease epidemically and endemically under similar conditions of atmosphere, soil and temperature. This, however, does not necessarily imply that the essential cause of the two is the same, and that each affection is a different manifestation of one and the same poison. Indeed the very contrary is known to be the case, despite the intermittency of type which may be assumed by the intestinal affection. Sullivan, speaking of tropical dysentery, says that it and intermittent fever may exist separately or conjointly, and that both are products of specific emanations from the soil, distinct in their cause as well as in their effect; but that, although the peculiarities of the soil in which the malarial fever is generated are known, we are unable to identify the nature of the soil peculiar to endemic dysentery. And again, to this poisonous infection due to locality, dysentery owes its endemic nature which distinguishes it from sporadic and epidemic dysentery of temperate climates. Be this as it may, there seems to be a noxious something peculiar to the place, which acts, either as a means of development and conveyance of the specific cause, or by increasing the susceptibility of the human organism to its influence, or perhaps both. The preceding remarks concerning endemic dysentery of hot climates may be repeated in detail and upheld as in the main applicable to endemic dysentery of temperate climes, endemic simply implying the persistence of and limitation to a definite area of the cause, known or unknown, of a disease.

Malaria.

Prior to and at the outset of sanitary science, when the importance of sub-soil drainage and the proper cultivation of the land was only just beginning to be intelligently understood and appreciated, dysentery and malaria were in this country often endemic together and confined to certain

definite tracts of country, and sometimes in the case of the former limited even in towns to well-defined areas. In the outbreak of dysentery in Cork, 1818-19, two forms of dysentery were described—one connected with typhus fever, epidemic and contagious; another with intermittent fever, endemic and non-contagious. With, however, improved drainage and cultivation of the soil, malaria may in this country be said to have practically disappeared, and dysentery—excluding the outbreaks from time to time in prisons and asylums—may be said to consist chiefly of sporadic or imported cases. Summing up, therefore, concerning its association with malaria and typhus, we find, on examining into the circumstances under which it arises, that dysentery may occur in connection with other diseases endemic and epidemic, with them may exhibit these two properties and have factors in their causation common to the co-existing affections.

With the recognition and removal of the causative elements, the diseases in question tended to disappear, till in the present day there are many who have no practical acquaintance with any of them. What, however, may be the specific organised agent in dysentery is as yet undecided.

From a clinical point of view there would seem to be no real distinction between tropical dysentery and the disease as it occurs in temperate climates. In both, the severe and mild types of the disease, endemic and epidemic, present pretty much the same onset, symptoms and progress; and anatomically the plates of the inflamed and ulcerated bowel, from cases here, show ravages not to be exceeded in extent and destructiveness by the most virulent tropical form. Would anyone pronounce, after a description of the symptoms and morbid anatomy of the cases—hereafter to be detailed—as to the tropical character or not of the disease, or doubt its being dysentery? That dysentery has its abode specially in the Tropics is undoubted; and that it has in the past existed in this country epidemically

and endemically, and that it may and does occur in the present with all its former characteristics, notwithstanding the absence of a specific emanation from the soil, merely shows that it is capable of developing under the most varying circumstances. The meaning, therefore, attaching to endemicity, as applied to the disease in the Tropics, only indicates the existence of a cause limited as to locality, and which is in every probability—as has been shown in other countries also at one time the habitat of dysentery and malaria—capable of removal by drainage and cultivation. The difference between the disease, then, in the two zones, seems more apparent than real, and certainly impartial observation does not warrant any sharp line of demarcation being drawn. Tubercular disease, leprosy, enteric fever remain the same diseases irrespective of their location, and why not dysentery?

Phthisis Pulmonalis.

Turning now to the association of dysentery with tuberculous disease of the lungs, one might naturally expect that the former would have an easy choice of victims in those suffering from phthisis. Contrary, however, to preconceived ideas, a combination of the two diseases is not so common as at first might be supposed; but that it does occur, and adds greatly to the difficulties of nursing, and to the distress of the patient, I have often enough observed. Since, however, the erection here of a consumptive hospital for females, with its better equipment and facilities for prolonged indulgence in open air and sunshine, the combination of the two diseases, at least so far as the women are concerned, is rarer. When the lung affection is progressing, the supervention of dysentery speedily produces a fatal issue. If, on the other hand, the phthisis is stationary or chronic, and unaccompanied at the time by any intestinal or gastric disturbance, dysentery, if it supervene, does not as a rule exhibit its usual severity, at least as regards its effects on the constitution of the patient, but tends to become chronic. The best example I have been

able to furnish of chronic dysentery in a case of chronic phthisis is that of Elizabeth C——, a coloured plate of whose bowel is presented.

As a rule, then, we may say that dysenteric ulceration of the large bowel seldom occurs if the small intestine is already the seat of well-marked tubercular disease; and, similarly, tubercular disease of the small gut does not often supervene when the patient is the subject of dysentery. In other words, the two diseases are seldom found co-existent *post-mortem*. From a large number of *post-mortem* examinations of consumptives, I have only been able to pick out about a dozen which exhibited, either with or without tubercular disease of the small intestine, indubitable evidence of dysenteric inflammation and ulceration of the colon. Generally speaking, as a result of my observations, those suffering from tubercular disease of the lungs seem to have a peculiar immunity from dysentery, even though living in the same ward with, and exposed to the same possible sources of infection as the latter. These assertions I have based on both clinical and *post-mortem* evidence. A glance at Plate No. IV. will convince that the ulceration of the colon, although occurring in a patient with chronic phthisis and associated with some small tubercular ulcers in the small intestine, is not tubercular. The other plates, all taken from cases free from any suspicion of tubercular disease, I have known to be almost exactly reproduced in consumptives, as in the comparatively few instances just recently mentioned.

Scurvy and Purpura.

Fortunately the combination with these affections, more especially scurvy, now almost never presents itself, though medical literature proves it to have been very common in long sea voyages, campaigns, sieges, etc. The conditions under which states of mal-nutrition, capable of inducing these diseases, arise, are so well known as to require only a brief mention on my part. The prolonged use of cheap fresh meat, as well as salted provisions; inferior flour and rice, bad potatoes; the influence of a moist, damp

and impure atmosphere in a confined and overcrowded space; prolonged confinement to bed and great mental depression; all separately or combined conduce, not only to its development but also to that of dysentery, and ofttimes in the same individual. In other words, it would seem that more or less similar causes, according to their duration and intensity, along with differences of constitution inherent in the individual, result in the development of one or other of the three diseases —purpura, scurvy and dysentery. So much has a close connection between these three affections been borne in on the minds of observers as to have given rise to the names, land scurvy for purpura and "scurvy of the bowels" for dysentery. That an intimate relationship does exist, is, I think, highly probable, and such a conclusion appears legitimate from the following:—

1. Their frequent occurrence at the same time, separately or in combination. In 1846-47 in Ireland, during the potato famine and when vegetables generally were difficult to procure, the three diseases were remarkably prevalent.

2. Their development under almost similar circumstances and corresponding disappearance with removal of these.

3. A general similarity to be observed in their signs and symptoms, and in the peculiar cachectic condition which they induce.

4. Two of them are specially characterised by the occurrence of hæmorrhages and ecchymoses dependent upon the same pathological cause—rupture of the vessel walls.

Although I have stated that the appearance of well-marked scurvy in asylums and workhouses is now an event of extreme rarity, still I have occasionally observed it, and I am firmly convinced that purpuric and scorbutic taints are more frequent among the insane than is generally supposed. The only class of cases in which this is at all likely to occur is in the feeble and perhaps for years bedridden patients, who, owing to inability to masticate, or an unwillingness—due to dislike of flesh diet—to partake of meat, have lived almost

exclusively on a boiled-bread-and-milk regimen. The recognition of this possible contingency is, however, usually sufficient to prevent its occurrence, which may generally be attributed to some oversight in the matter of dietary, or the presence of some profound deterioration of the blood and secretions of the insane of which we are ignorant.

CHAPTER III

CLINICAL HISTORY, SYMPTOMS, TREATMENT, ILLUSTRATIVE CASES, ETC.

Definition and Classification.

It would seem only natural to expect that a disease known and studied from antiquity should have many and varied classifications, according as writers viewed it from a clinical, pathological or ætiological aspect. This may also be readily understood when we have regard to the different characters it may assume, according to the prominence of this or that group of symptoms in individual cases, to the various forms it presents in different epidemics, and to the association or complication with other diseases such as malaria, scurvy and typhus.

The terms dysentery, colitis, colonitis, difficultas intestinorum, rheumatism of the intestines, abdominal coryza are a few of the names given to the disease, which, without attempting anything like a complete and exhaustive definition, may be designated as a specific primary colitis, characterised by ulceration and sloughing of the mucous membrane; by more or less intractable diarrhœa, the stools containing and often consisting entirely of blood-stained mucus and in the later stages of the disease becoming very offensive and possibly containing sloughs and shreds of membrane; by a varying degree of fever; and by abdominal pain and uneasiness, with in many cases tenesmus and strangury.

In the classification about to be adopted, and to guard against any misinterpretation, it is to be noted that ulcerative colitis occurs, in both non-specific and specific affections, other

than as dysentery, which with its varieties are alone comprised in the first group. When present, however, in specific diseases, the ulceration of the colon is to be looked upon as only a more extensive manifestation of the said diseases and not as an independent and primary affection originating in and limited, as in the case of dysentery, entirely to the colon.

Exceptions nevertheless do occasionally occur — one of which has already been alluded to in discussing the association of dysentery with phthisis pulmonalis.

The following classification will perhaps more clearly convey what I have attempted to explain in the text.

According to it, cases of ulcerative colitis are divided into two groups :—

1. Idiopathic ulcerative colitis.

   By this is meant a specific affection (due to micro-organisms differing according to the climate in which it occurs, or perhaps different methods of investigation) beginning in, and throughout its course almost invariably limited to, the large bowel. The only disease which appears to fulfil these requirements, and which answers to the definition already given, is dysentery. It and its varieties are therefore alone included under this heading.

2. Secondary ulcerative colitis.

   (*a*) Specific, in the sense that it is due to a specific poison and occurs in the course of a specific disease, either as a more extensive manifestation of the disease of which it forms a part, not as a specific disease originating in and confined to the colon, or as a comparatively rare complication.

   Examples of implication of the colon in this sense are to be seen in colo-typhoid, tubercular and syphilitic disease, diphtheria, measles, etc.

   (*b*) Non-specific, as occurs in impaction of fæces,

presence of foreign bodies, and in toxic and irritant poisoning from diseased food, corrosive sublimate, uræmia, etc. In the two sub-divisions of this group it is to be observed that the small intestine is almost in every instance involved, and that more extensively than the colon. Exceptions are, however, to be noted in the cases of syphilitic and tubercular ulceration of the rectum, where the small intestine may be perfectly healthy, and in the case of typhoid fever in the old, where Peyer's patches are atrophied.

As these conditions do not, however, enter into the scope of this work, only passing mention is made of them. Further, in the case of toxic and irritant poisoning, other parts of the gastro-intestinal tract are involved; but as in these cases there is nothing of a specific or idiopathic origin, and the clinical signs and symptoms are of themselves sufficiently diagnostic, no further inquiry in this connection is necessary.

Referring now for a brief space to the terms employed to denote inflammatory conditions of the different divisions of the gastro-intestinal tract, it is imperative for a clear conception of the sense in which they are used—if we are to avoid misunderstanding and confusion of nomenclature—that a definite and uniform meaning be attached to each.

As expressive of inflammatory conditions of the intestines we are familiar with the terms gastritis, enteritis and colitis, and their combinations gastro-enteritis and entero-colitis. No confusion attaches to the employment of the first and third of these names—their meaning is sufficiently conveyed by the derivation. The same, however, cannot be said regarding the use of the word enteritis, it being not sufficiently clear what precisely is meant.

Hamilton in his *Text Book of Pathology* tabulates croupous enteritis as synonymous with English dysentery, regarding

which he says, "The large intestine is the part of the bowel usually said to be affected with this disease."

Fagge avoids the use of the term enteritis, and says there is no distinct affection requiring to be described under that name, and indicates that possible cases would clinically fall under the head of severe diarrhœa or dysentery. "To give an account of enteritis as a substantive disease is to deal over again with cases which have to be fully considered under other heads."

Without questioning the views of such an eminent authority as to the non-existence of such an affection, I think that the term, as used in the combinations gastro-enteritis and entero-colitis, serves a distinctively useful purpose, both clinically and anatomically, as indicating the participation in the inflammatory process of the small intestine.

Strümpell, although he defines dysentery as a disease of, and practically limited to, the large bowel, includes under the designation catarrhal enteritis, affections in which, along with the small intestine, the stomach or colon must obviously be involved, and that in some instances to a greater extent.

Ziegler also appears to include under the general name of intestinal catarrh or enteritis inflammatory affections of different portions of the gastro-intestinal tract, admitting, however, that these have been variously termed, according to the particular part affected.

The same objection may be urged to the inclusion by Boyce in this term, of affections in which the colon and small intestine are separately or together implicated.

In *Quain's Dictionary of Medicine* it is stated that, as a rule, the term enteritis is restricted to inflammation of the small intestines.

I have quoted these authorities to show the latitude allowed in the application of this term, and to indicate the difficulties it presents to the statistician who has little or no guide as to the exact affection it is intended to indicate.

Reverting again to the classification of our subject, we

distinguish between epidemic, endemic and sporadic dysentery. It is, however, not the intention of the writer to discuss this aspect of the disease, and beyond stating that here, as in other asylums, it may be regarded as endemic, with occasional increments of epidemic severity, no further allusion in this connection will be made. For clinical purposes, however, it is necessary to distinguish between different cases, and the division which seems most to accord with those that have come under my observation is that adopted by Frank and Horn, namely, acute sthenic, acute asthenic and chronic. Though not perhaps a strictly scientific one, it appears the most suitable, considering the class of patients in whom the disease occurs. The onset and symptoms I shall describe generally, indicating, according to the presence or special predominance of this or that symptom, the differences to be noted in the two types of acute cases. These differences, however, I may here state, are chiefly to be found in the temperature and general condition of the patient, and the duration and mortality, and will perhaps be more clearly understood by a reference to the illustrative cases.

Concerning its onset, it is, from the class of patients we have to deal with, often very difficult, and at times impossible, to obtain reliable information. Attention is thus usually first attracted by the occurrence of diarrhœa, which, from bitter experience, we always look upon as prospective dysentery and take our precautions accordingly. In many cases the purging may only be slight, caused by some indiscretion in diet, and might easily have been treated in the ordinary wards; but, for the reason already given, unless the patient is fairly intelligent and in good bodily health at the time, I prefer, when it is possible, to send him to the hospital. This ensures early observation, and the employment of active treatment from the beginning, and, if it turn out simple diarrhœa, the gain still remains ours. It is otherwise almost impossible in a large ward to obtain accurate information as to the number and character of the motions.

Onset.

The appearances presented by the evacuations in dysentery are sufficiently well known to render it superfluous on my part to describe them in anything like detail. From admixture of fæces with blood and mucus to the meat washings, or even, as often enough happens, to the discharge of a puriform fluid the colour of cream, and with a most horrible fetid odour, all have been observed and are characteristic of the cases here. Scybalæ I have seldom seen passed in the course of the attack, and have never observed them present in the bowel post-mortem. Indeed, the very contrary seems to hold, the contents of the gut being very fluid and scanty. There is usually an early appearance of tenacious mucus which clings to the bottom of the vessel, and which may be either pure and present an appearance like partially dissolved gum arabic, or may be more or less intimately mixed with blood, from mere staining to a condition recalling the rusty sputum of pneumonia. This occurs in both sthenic and asthenic cases; but in the latter, from almost the onset, and in chronic cases, I have not infrequently observed the dejecta present a pale diarrhœal appearance which, in some asthenic cases, towards a fatal termination becomes almost puriform. The odour in such cases is of itself sufficiently characteristic to warrant a diagnosis.

Dejecta, appearances of

The attack is not uncommonly preceded by bilious vomiting and pain in the belly, though the latter is often, even among fairly intelligent patients, little complained of even on pressure. Tenesmus may or may not be present, and is often slight. Here let me mention the indifference and non-sensitiveness to bodily suffering frequently exhibited by the insane; and, bearing this in mind, we must not therefore expect the moans and other evidences of intense suffering and distress which we would naturally look for as playing a prominent part in others.

Vomiting, pain, tenesmus

Rigidity of the abdominal muscles is also a feature of the complaint not often observed in the insane; and from the extensive damage inflicted on both the mucous and muscular

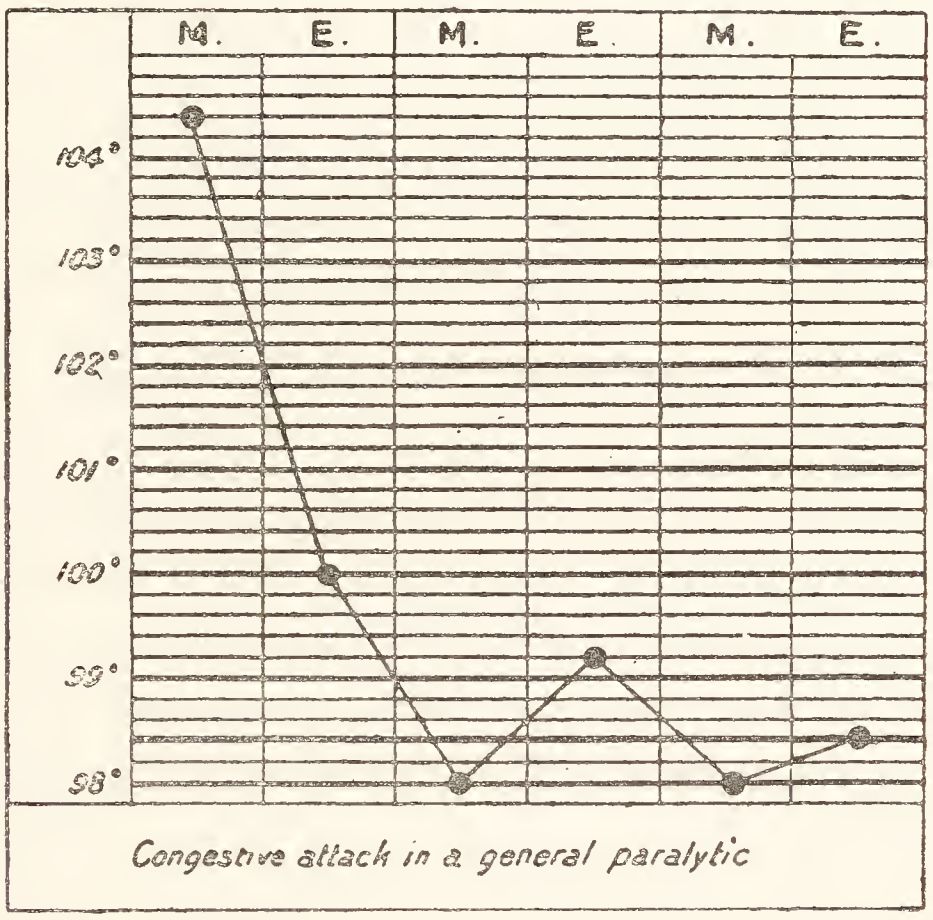
coats of the colon one would expect the peritoneum to be involved much more frequently than it is. The absence of tormina and other signs of abdominal discomfort may perhaps be explained by the specific poison and vitiated intestinal secretions, exercising a benumbing influence on the exposed sensitive nerve fibres. Be this as it may, the fact remains that symptoms and signs which are very pronounced in ulceration of the colon in the sane are here more or less conspicuous by their absence—a possible explanation of which has been ventured.

Rigor and Temperature.

The onset may also be characterised by a rigor and rapid rise of temperature, up to even 105° F., with hot, dry skin, or occasionally profuse perspiration, and a dry, parched tongue. Just let me mention in passing the intensely disagreeable, and I might almost say characteristic exhalation from the cutaneous surface frequently observed, especially in the asthenic cases where the surface is often clammy and moist. Generally speaking, a rapid and extensive rise of temperature is met with only in the acute sthenic cases, the asthenic type as often as not running its course with only a moderate degree of pyrexia, or being even afebrile. I have at present before me a number of temperature charts from acute cases of both types, and in addition to that of range, one is struck by the enormous sudden and early morning remission met with in the sthenic forms, amounting in some instances to more than five degrees, and this even without the aid of antipyretics. This is a circumstance I have only observed in the following class of affections, congestive attacks of general paralysis, influenza, ague and urethral fever. The accompanying woodcuts illustrate—with the exception of one of ague—the temperature as it frequently occurs in the above. This rapid fall may, so far as dysentery is concerned, be regarded as of good omen, the majority of cases exhibiting it usually terminating favourably. On the other hand, where the morning remission does not take place, or has been insignificant, and the temperature only falls to

normal after several days, or where, when never even higher than 102° F., it remains so for more than a week, the prognosis must be much more guarded. Of six cases under my own immediate observation last year which presented this extensive remission, all made a good recovery.

Another feature presented by the temperature, especially of the more prolonged and asthenic cases, is the irregular oscillation, resembling in measure what one often sees in tubercular disease of the lungs and in suppurative affections.



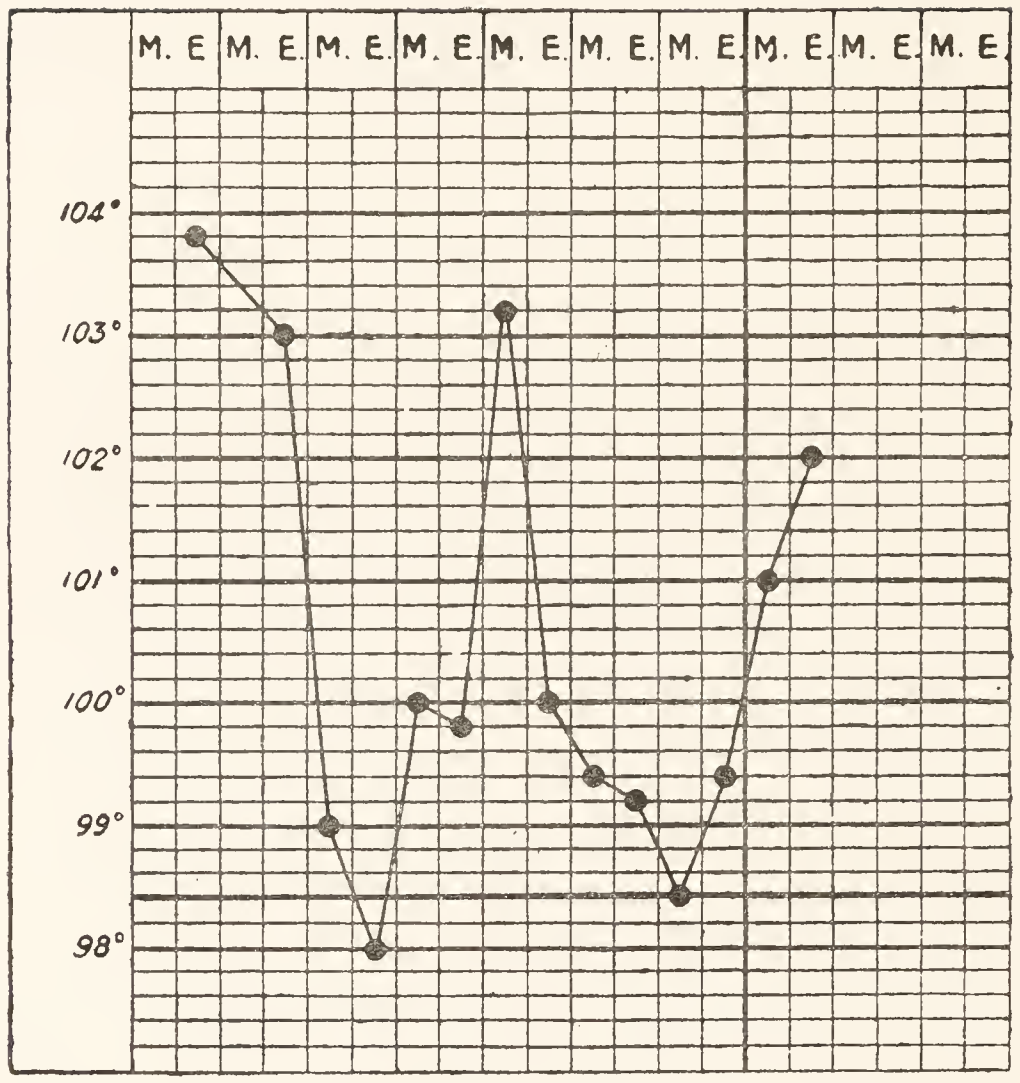
Congestive attack in a general paralytic

Pulse.

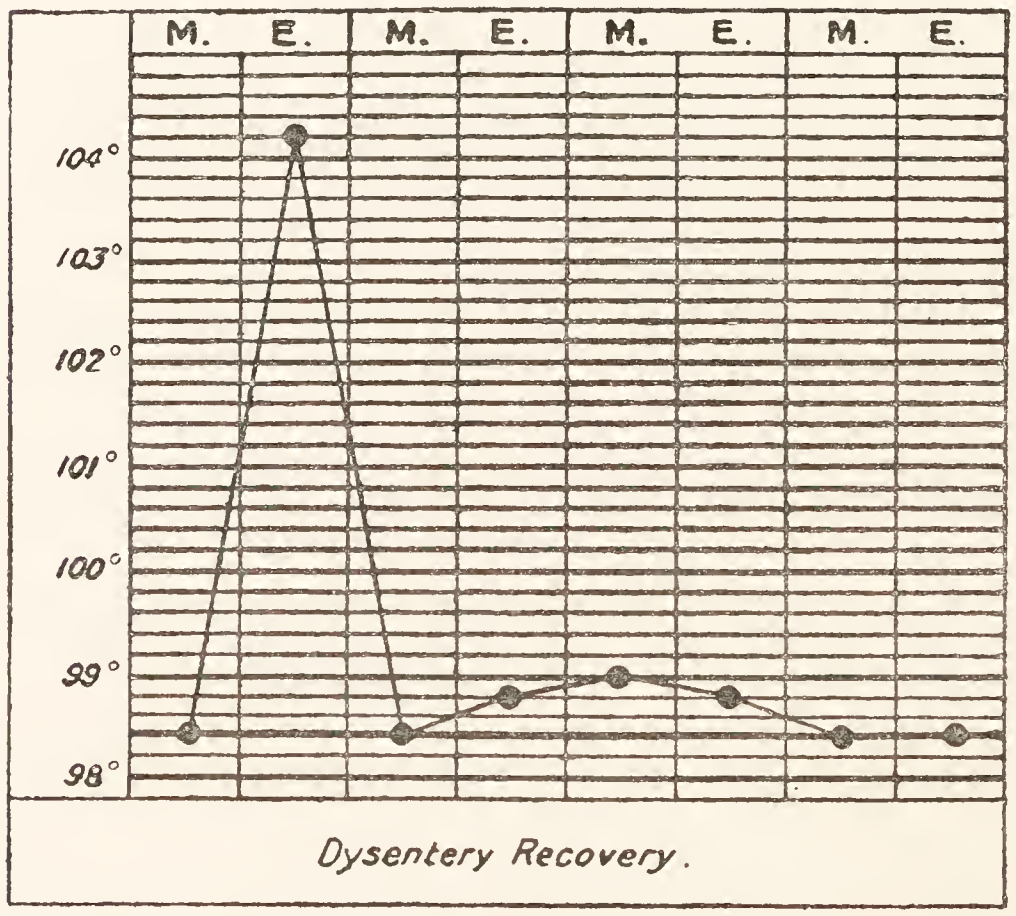
The pulse presents nothing specially to be noted, its rate corresponding usually to the degree of fever, and its strength varying according to the condition of the patient.

Tongue.

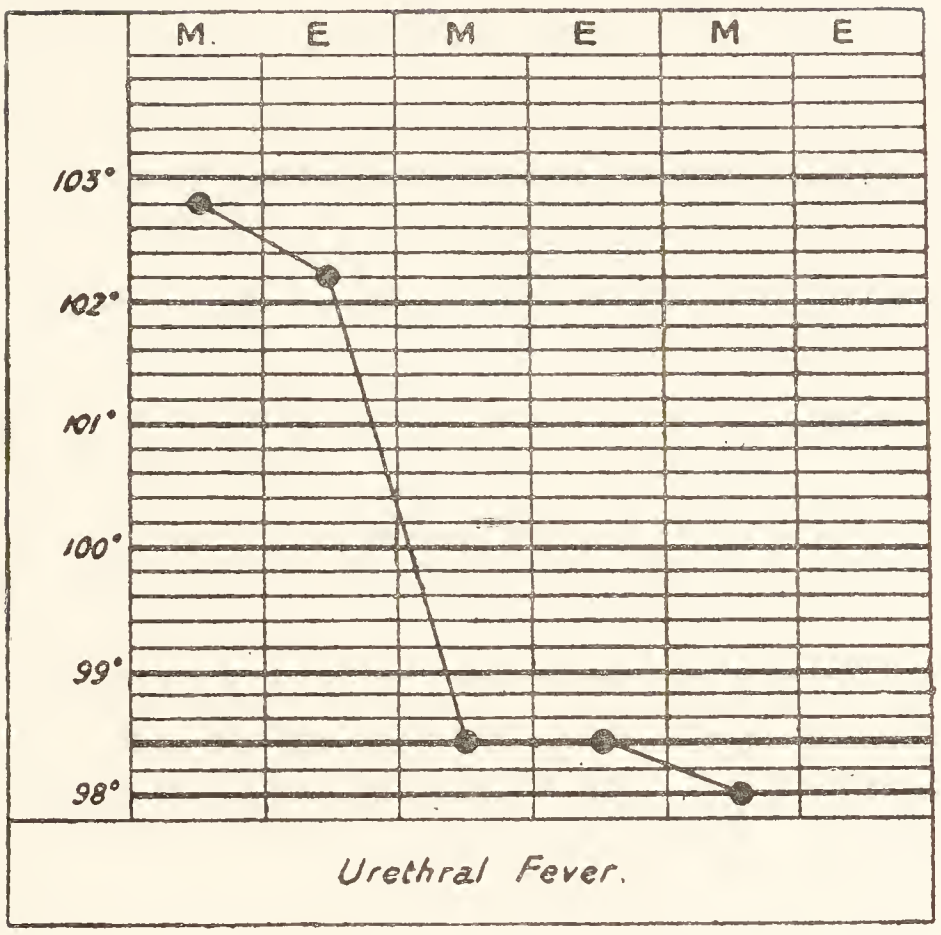
The tongue, more especially in the acute sthenic cases, is dry, clean, red and glazed. In asthenic cases, it is at first moist and covered with a thin, whitish fur. As the disease progresses, it becomes more thickly coated, dry and cracked, and when a fatal issue is to be apprehended, covered with sticky mucus and dirty yellowish



Dysentery Fatal



Dysentery Recovery.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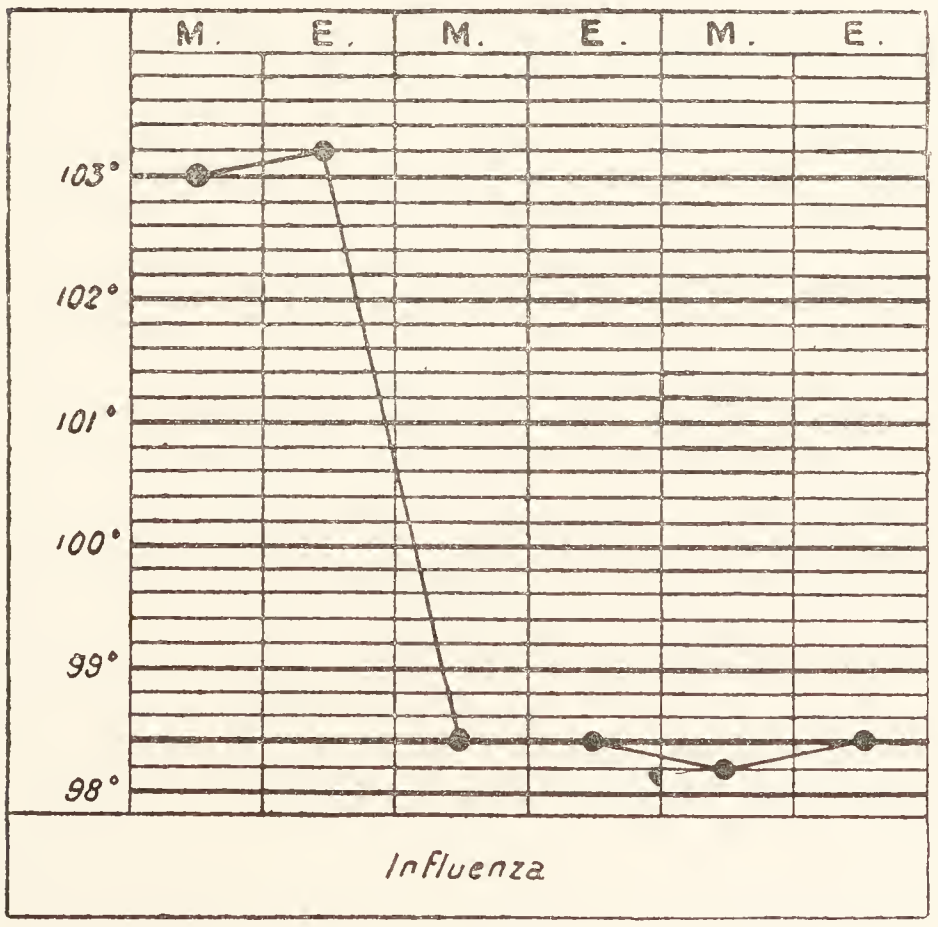
Urethral Fever.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101°
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99°
98°
Influenza

D

brown crusts. The lips, teeth and palate in such cases become coated with sordes, the gums show a tendency to ooze, the breath is most offensive and the patient unable to articulate.

Thirst.

Extreme thirst, with a sensation of dryness of the throat, is at times a most distressing symptom. The more intelligent of the patients also frequently complain of a very bitter taste in the mouth.

Urine.

Except in the cases attended by high temperature, when it is deeply coloured and loaded with urates and has a pungent, offensive odour, the urine presents nothing remarkable. Albumen is not often detected, and when present is usually accounted for by renal mischief; occasionally constituents of the bile are present.

Delirium.

Acute delirium I have never observed, even when the temperature would lead one to expect it. Towards the fatal termination, however, stupor and coma are not infrequent.

Duration.

This, in fatal cases taken individually, varies from a minimum of less than two days up to a maximum of over five weeks. Generally speaking, all much over a fortnight in duration belong to the asthenic type. Calculated upon forty-two fatal acute cases of both types, in which the duration was accurately determined, the average maximum and minimum in days is shown in the accompanying table, as is also the duration of eighteen acute cases ending in recovery, and eight chronic cases with a fatal termination. The general impression conveyed by the table is, that whereas it appears to be more rapidly fatal in the case of women, the recovery in the case of men seems slightly slower.

Duration of Acute Attack in Forty-two Fatal Cases.

Fourteen men and twenty-eight women.

MEN.		WOMEN.	
Average . . .	13 days	Average . . .	10 days
Max. . . .	21 ,,	Max. . . .	25 ,,
Min. . . .	6 ,,	Min. . . .	3 ,,

Duration in eighteen acute cases ending in recovery.

MEN.		WOMEN.	
Average . . .	8 days	Average . . .	7 days
Max. . . .	18 ,,	Max. . . .	16 ,,
Min. . . .	4 ,,	Min. . . .	4 ,,

Duration of eight fatal chronic cases.

MEN.		WOMEN.	
Average . .	months 5 weeks 2	Average . .	months 4 weeks 1
Max. . .	,, 10 ,, 0	Max. . .	,, 11 ,, 0
Min. . .	,, 2 ,, 0	Min. .	,, 1 ,, 2

Note.—The case of Wm. M., which was fatal within two days, is not taken into the above calculations.

Chronic Dysentery.

These cases are usually chronic from the outset, and only occasionally result from the acute forms. They commence

as an intractable diarrhœa, continuous, or interrupted by intervals of varying duration, during which the stools may present a fairly normal appearance. Some patients are purged daily for many months, and in the end die of exhaustion or some intercurrent affection. Others again, with considerable intervals of freedom, are more or less subject to it for years. These latter, however, must not be confounded with those having second and third attacks, in which the interval is much more prolonged, and the patient in the enjoyment of good health and often gaining in weight. The appetite usually remains unimpaired, and if the purging is not excessive, the patient may appear fairly healthy. This, however, is the exception, the subjects of chronic dysentery being usually thin and emaciated, living skeletons in fact, and the wonder is how they survive as they do. There is rarely straining at stool, and only towards the end loss of control over the sphincters. The dejecta are fluid, scanty, for the most part feculent, though at times containing bloodstained mucus, and have a most offensive odour. The more intelligent of the patients often complain of a pain in the small of the back—rheumatism they call it. The skin is harsh and dry, and the tongue, as a rule, clean.

Complications.

Of all complications, implication of the lungs is the most frequent, and is almost invariably fatal. I exclude here all chronic or pre-existing pulmonary affections, and include only those arising during the course of the bowel complaint. The one which presents itself with greatest frequency is congestion and œdema, after which the most common is pneumonia—croupous and catarrhal. This is always fatal in my experience, and whenever anything approaching the signs of consolidation can be detected, I consider the case as hopeless. The part which cardiac disease

Pneumonia

plays calls, I think, for no special remarks; the same considerations are to be taken into account here as in any other acute disease in which it may exist.

If one takes a healthy condition of the urine as a guarantee of a sound condition of the kidneys, renal disease is comparatively uncommon, and at best can only be regarded as accidental. Although cirrhotic changes figure somewhat largely in the post-mortem statistics, many of them I am inclined to regard as actual or premature senile changes.

Kidney Disease

General peritonitis, with or without perforation, is very rare, and in the recorded post-mortems has only been found once. Of equal rarity seems to be localised peritonitis from contiguity, with adhesion of neighbouring viscera and coils of intestines. Note in connection with these complications the very opposite condition obtaining in tubercular disease of the bowel.

Peritonitis and Perforation

Abscesses of the glands and viscera, considering the favourable opportunities for their occurrence, are comparatively uncommon, and in only two instances was abscess of the liver found post-mortem. Perhaps the absence of malaria and high modes of living have something to do with this, the hepatic tissue under such circumstances not proving very vulnerable.

Metastatic abscesses

Prolapsus ani and retention of urine alone, or in combination, are not infrequent. In two instances I found them associated with extensive sloughing of the rectal mucous membrane. Unless great care is exercised, the ordinary bedsore is, from the acrid nature of the evacuations, combined with in many cases the patient's utter neglect of cleanliness, very apt to occur. Trophic bedsores are comparatively rare, and the result of some pre-existing nervous affection. Needless to say, their appearance is invariably fatal.

Prolapsus ani, &c.

Bedsores

Contagion.

And now a few words on this subject. As may be expected where dubiety exists, the assertions for and against are equally positive; one group of observers insisting upon the possession of this property, another as firmly denying it. It is apparently not inoculable, if we accept as satisfactory evidence the means adopted by one surgeon of using an uncleansed enema syringe on those free from the disease. This, so far as I am aware, is the only direct experiment on man. Some, however, state they have seen it communicated in this way, and others consider it to have been caused by the infected air of a night chair acting on the extruded mucous membrane of the anus.

In Chapter II., under the heading of association with malaria, I have mentioned that two forms of dysentery were described, one associated with intermittent fever endemic and non-contagious, another with typhus epidemic and contagious. Its combination with the latter certainly threw obvious difficulties in the way of gauging and accurately determining its possession of this property. Many of the earlier writers held that the asthenic varieties consisted of dysentery complicated with typhus fever, and that it is the latter which is contagious. As has, however, been pertinently asked, why was it always the dysentery and not the fever that was propagated, and why was this contagious dysentery often present where there was not even a solitary case of fever? Considering, however, the circumstances under which the bowel complaint arose, it is difficult to determine whether its spread was not due to a continuance of these conditions, and not to a contagious element inherent in the disease. This may be offered as a possible explanation of the discrepancies existing in the different accounts on the subject. Personally, I am aware of only a solitary instance in which it attacked those attendant upon patients suffering from it. Patients employed in emptying earth

closets—where these are in use—are said to be peculiarly liable to be attacked. Knowing the degraded habits of some, and the general carelessness as to cleanliness of the hands exhibited by many of the insane, a possible means of direct infection by the fingers through the nostrils and mouth is thus suggested. This is rendered still more likely by the circumstance that attendants or cleanly patients employed in the disposal of the dejecta are not specially singled out for attack. The eating of excrement, so often indulged in by certain patients, is often ascribed as the source of infection, but I do not think that much value is to be attached to the assertion.

Diagnosis.

The recognition of dysentery—leaving cholera out of the question so far as this country is concerned—does not, as a rule, present much difficulty. The character and peculiar odour of the evacuations, the frequent purging, the rapid and great prostration, in many cases the temperature, and in general its resistance to ordinary methods of treatment, are the chief points which serve to distinguish it from ordinary diarrhœa. I may mention, however, that during outbreaks of dysentery, ordinary diarrhœa often becomes difficult to check, and does not seem to be affected by the usual run of remedies, and in particular by opium. In fact, generally speaking, diarrhœa among the insane is more difficult to control than the same affection occurring in general and hospital practice.

Certain diseases, some of which belong rather to the domain of surgery than medicine, may be and have been mistaken for dysentery, and to these, and the salient points of distinction between them and dysentery, it is necessary for a brief space to direct attention. Enteric fever, acute intussusception, acute gastro-enteritis, malignant disease of the rectum or sigmoid flexure and proctitis include the affections most commonly mistaken for dysentery.

Beyond stating that in the epidemics of the beginning

and middle of the present century, owing to the confusion which then existed concerning enteric fever, the post-mortem records of dysentery contained many cases which should perhaps have been more correctly classed as that affection, no special reference will be made to the points of difference between these two diseases. I would, however, point out that in the elderly and aged, owing to the atrophy and disappearance of Peyer's patches, the morbid intestinal processes in enteric fever are most marked in the colon, the small intestine, with the exception perhaps of slight congestion, presenting a healthy appearance. When such persons are attacked by enteric, a certain amount of confusion may arise owing to the lesions sometimes resembling those of dysentery. Anatomically it is in some cases almost impossible to differentiate. A close attention, however, to the mode of onset and progress, general condition of the patient throughout the disease and the temperature, apart from the discovery of Eberth's bacillus, will usually serve to distinguish enteric fever. Of course, it is quite possible for dysentery to complicate enteric fever, but I would be very chary of pronouncing any case as dysentery where Peyer's patches were the seat of ulceration. Rather would I prefer to ground my opinion as to the presence of this complication on the clinical, not the pathological phenomena. The more pronounced lesions of and sometimes limitation to the colon in cases of enteric fever in the elderly and aged, may be attributed to the slower atrophy of the solitary follicles of the large as compared with those of the small bowel; indeed, it may be questioned if they ever in the former totally disappear, not at least till very late in life.

In acute gastro-enteritis there is usually a history obtainable of the ingestion of some irritant, and where this is wanting, the sudden onset of symptoms indicative chiefly of involvement of the upper reaches of the intestinal tract, and in many cases a collapse temperature are sufficient to distinguish.

In acute intussusception the occurrence of tenesmus and diarrhœa, along with blood and mucus, presents a group of symptoms more liable than gastro-enteritis to be mistaken for dysentery. The greater frequency of vomiting, its persistence, its subsequent feculent character, and the early and greater collapse are characteristic of this form of intestinal obstruction, and point to the diagnosis. Digital exploration and the use of the speculum and bougie, along with the history of the case, will determine the presence of malignant disease. The comparative absence of constitutional symptoms, its usually much shorter duration, the passing of formed or partially formed fæces, and examination by means of the finger and speculum, will in the main distinguish proctitis, fissure and ulcer of the rectum from dysentery. Dysenteric proctitis is a name sometimes used to indicate that the specific inflammation is entirely confined to the rectum. Of this I can recall one notable example in which I removed at different times almost the entire mucous membrane, which was in a sloughing condition.

And now, having disposed of the affections which may be confounded with dysentery and the means employed to distinguish them, let us turn our attention to the distinction, if any, which exists between dysentery and the so-called simple ulcerative colitis. I may premise at the outset that I do not agree with those who recognise in this condition of the colon a simple colitis distinguishable from dysentery; indeed, there is strong evidence to adduce that it is a specific affection, due to a definite micro-organism, identical with one to which the origin of dysentery is attributed. An account of this bacterium and the methods employed in its isolation and cultivation will be found in the chapter dealing with the ætiology of dysentery, for the details of which I am indebted to Dr Goodliffe. Meanwhile I will try as clearly as I can to explain my views as to the nature of this simple ulcerative colitis, and why I regard a disease prevalent in asylums, having so much the characters of a specific affection, as not

differing from but being in very truth dysentery. That most indefatigable writer, the late Dr Fagge, does not mention it in his *Text Book of Medicine* as a distinct disease; indeed, so far as I can perceive, he seems specially to have ignored it. This from a man of his standing would seem to imply that he either did not recognise its existence, or else classed it under another name. The latter supposition is, I think, the more likely. Under the heading of diagnosis in the chapters on dysentery and enteric fever, he records in the former two well marked instances of sporadic dysentery with very extensive *diffused ulceration of the large intestine*, the presence of which had been entirely unsuspected during life; in the latter he says, "Two or three instances have occurred in Guy's Hospital in which *acute diffused inflammation of the colon*, generally of a diphtheritic character, has been mistaken for enteric fever, there having been no symptoms of dysentery, so far as could be afterwards ascertained."

And referring to this mistaken diagnosis of enteric fever he says, "The characteristic symptoms were absent, or passed under the name of diarrhœa." Observe that in both instances he speaks of "the diffused ulceration" and "the acute diffused inflammation of the colon" not as simple ulcerative colitis, but as dysentery. This, together with the perusal of Dr Hale White's article on simple ulcerative colitis in Guy's Hospital Reports, leads me to believe that these two authors did not perhaps take the same view of a similar disease, the latter classifying under the title of simple ulcerative colitis, what the ormer would have called dysentery. Most medical writers either do not mention or only very cursorily describe ulcerative colitis, apart from its occurrence during the course of other diseases, in which it gives rise to what are termed secondary dysenteries. It may be gathered, then, from the foregoing that the literature on the subject is not extensive, and to Dr Hale White may, I think, be attributed the credit of being the most prominent in claiming for it a position as a definite disease distinct from dysentery. To his articles, therefore, I

would direct attention ; and, apart from the interest attaching to anything from his pen, it is essential, in order to convince that what he describes as simple ulcerative colitis is in reality dysentery, that a careful and undivided attention be paid to the illustrative cases from which he draws his conclusions, and to what he asserts are the distinguishing features between it and dysentery.

In the *Lancet*, 10th December 1887, he describes a case of ulcerative colitis, and says this condition is rare, its origin unknown or vague, cause unknown, appears to be invariably fatal, cannot be detected during life with any certainty, and is certainly distinct from dysentery. In a pamphlet on simple ulcerative colitis and other rare intestinal ulcers by the same author, case 1—which for all practical purposes may be taken *plus* the clinical history as similar to the one he records in the *Lancet*—is described as having suffered from diarrhœa, vomiting and abdominal pain three weeks, and with blood in the stools two weeks, before admission. The dejecta are described on admission as almost liquid, containing clots of blood, a little mucus and some fæces. Stress is laid upon the motions not being dysenteric. Observe that the disease was well advanced before the patient came under observation in the hospital, and that the description of the evacuations, which might have been typically dysenteric at the outset, was taken apparently from the patient's own statements. Further, one would not expect to find, after three weeks' duration, much, if any, mucus in the stools in a case of dysentery; in fact, it would be the exception, large areas of the mucous lining of the bowel having by this time disappeared, and healthy portions with power to secrete being few and far between. The autopsy revealed universal ulceration, leaving, however, irregularly shaped islets of mucous membrane, edges of ulcers sharply defined and muscular fibres exposed. This answers in almost perfect detail to *post-mortem* records of cases of epidemic dysentery in the present century. I have selected this case because it appears to me to be one of the most typical of the

series of eleven cases he instances, and to serve as a basis upon which to found my reasons for dissenting from the author. Several of the others, however, occupy rather dubiously, I think, a place in the series, concerning which opinion would not be unanimous. There will, however, only be mentioned Nos. 4, 8, 9 and 11, Nos. 8 and 11 being the subjects of purpura.

Let us see now what Dr Hale White considers the main features of simple ulcerative colitis, and how he proposes to distinguish it from dysentery. Its rarity and vagueness of origin have already been mentioned in the case quoted, and the pathological lesions—in some cases—are admitted to be indistinguishable from those of dysentery, a definite specific disease with definite symptoms, which characteristics, I claim, belong equally to simple ulcerative colitis, admitting for the time being that it is different from dysentery. We cannot as asylum physicians say that this ulcerative affection of the colon is rare, at least among the class of patients in which our practice lies. We have opportunities of observing it from the outset, and associate its occurrence with certain conditions already described. It is, moreover, strongly suspected to be occasioned by one of the micro-organisms to which dysentery is said to owe its origin, and it is attended with more or less definite symptoms and constitutional disturbance. A disease possessing these features we cannot, therefore, regard as simple.

So much for its alleged unknown origin and cause; and now let us see how it is proposed to distinguish it from dysentery. The rarity of acute dysentery is said to separate it from simple ulcerative colitis; but the reputed rarity of a disease is no reason for concluding that it does not occur. Beri-beri is also a rare disease, and yet it has been and still is prevalent in at least one asylum. In the article in *Fowler's Dictionary of Medicine*, 1890, simple ulcerative colitis is described as also a rare disease. If dysentery and ulcerative colitis, then, are both rare diseases, this peculiarity cannot be

of any diagnostic value. Wilks and Moxan admit in some cases the ulceration of the colon to be so identical with that found in dysentery that the diagnosis between the two depends on the constitutional disturbance. Are we to infer from this, that this extensive ulceration of the colon attended by such fatal results has no constitutional disturbance, or one at all events which will serve to distinguish it from dysentery? I am sure every asylum physician will bear me out when I say that the former is always attended by severe, often indeed most profound constitutional symptoms. Other distinguishing features between the two, upon which Dr Hale White relies, are the character of the stools and the prominence of tenesmus. The latter, I need not say, depends upon involvement of the rectum, and may thus be absent when this portion of the bowel is not attacked. Further, it is often slight in sporadic cases, and disappears or greatly diminishes in the later stages of even the most severe dysentery. Though marked tenesmus is not a prominent symptom in the bulk of our cases, still it is frequent enough to be noticeable, and a possible explanation of its absence has been offered, not inconsistent, I think, with what we know to be the case, and have often observed in other painful affections among the insane. In only two of Dr White's eleven recorded cases (Nos. 1 and 5) is any special mention made of the lower portion of the rectum being involved, and though tenesmus is not mentioned as being present, the motions are described as slimy and mixed with blood. Tenesmus, therefore, cannot be accepted as a distinguishing feature, as it may be present or absent in both dysentery and, what for the present we are considering as a different affection, simple ulcerative colitis. As to the character of the evacuations in dysentery, we know that they vary with the stage of the disease, which, when epidemic or endemic, presents every variety of dejecta, cases occurring in which throughout they are feculent. To quote Dr Fagge again, sporadic dysentery may exist unsuspected during life, and in three cases which he mentions it

was mistaken for enteric fever, the symptoms being absent or characterised by diarrhœa. Now, in none of the most typical of the eleven cases of simple ulcerative colitis were the patients under observation from the outset, but only after tenesmus and the characteristic appearance of dysenteric stools might be reasonably supposed to have passed away. If cases Nos. 1 and 5 occurred epidemically or endemically, what would be the diagnosis—dysentery or simple ulcerative colitis? Many of our cases here, and many recorded when dysentery was common in this country, present symptoms and pathological changes similar to Nos. 1 and 5. With all due deference, then, to Dr White, I would say of ulcerative colitis in asylums that it is—1. A specific disease endemic, epidemic, and there is no reason why it should not occasionally be met with sporadically in general and hospital practice. 2. It is probably due to a micro-organism, a bacillus, which, ingested with food, does not occasion the disease in rats. 3. It is fairly common, and arises under conditions similar to those with which dysentery takes origin. 4. Though a fatal disease, is still capable of cure in many instances. 5. That it is dysentery such as we know that disease to have been when endemic and epidemic in this country.

Treatment.

Prophylaxis.

Dealing at this juncture with the question of prevention, the chief indication consists in the assuring of perfect sanitary conditions, as implied by the proper ventilation of day-rooms and dormitories, and the placing of lavatories so that by no possible chance can the closet emanations penetrate into either. Under the same category I would also include arrangements permitting of the immediate removal of all soiled linen to a place specially provided, and if possible detached from the wards. During the night all dejecta in night-chairs, etc., should be at once disinfected, and if possible removed. Patients of dirty habits

should be confined to day-rooms and dormitories specially constructed for their reception, and well provided with baths for their immediate and thorough cleansing. The difficulties and increased expense in carrying out any such arrangements are obvious and weighty, and moreover, in many of the older asylums would—unless they were entirely rebuilt—be impossible of execution. In the planning of new asylums or of additional and detached blocks to existing ones, some such provision as suggested should be included. Further, the floors, especially of the hospitals and wards occupied by dirty patients, should be of pitch pine, and rubbed at least once a week with a mixture of wax and some essential oil. This variety of wood is not so absorbent, and does not so readily splinter as other kinds used in flooring, and if well laid down and properly waxed, offers little lodgment for dirt. Bedside carpets should also be dispensed with in the hospitals and wards referred to, as they simply serve to collect filth. In no case should there be more than fifty sleeping in a dormitory, and then only such as are of clean habits and able to attend to themselves. Those of filthy habits and the aged feeble—who are often, from affections incidental to their time of life, almost unavoidably so—should be placed in smaller dormitories. These general observations apply to the ordinary sleeping accommodation, but in the infirmaries a stricter limitation of unnecessary, I might almost say pernicious, furnishing is required. Here, in addition to the usual run of ailments met with in the wards of a general hospital, finally gravitate helpless dements and general paralytics, all more or less wet and dirty in their habits, and many with trophic bedsores.

Such occupants are eminently calculated to produce—especially where the ward is at all overcrowded—a condition of atmosphere not to be found in general hospitals, and undoubtedly handicap the recovery of others. How necessary, then, to have all superfluous articles of furniture and decoration strictly forbidden. The idea that a bright and

cheerful ward has a marked influence in hastening convalescence (I refer to medical ailments) is one that, as regards patients in general hospitals, cannot be disputed; but in the infirmary wards of an asylum, where the majority of the patients are of the class referred to, it cannot on sanitary grounds alone be advocated. Decoration here, therefore, becomes a matter of sentiment, and is altogether out of place. Why? Because the majority are far beyond appreciating it; and as for the intelligent patients who may happen to be temporary residents, sanitary considerations alone urge, even compel, that they should be deprived of the æsthetic indulgences often provided in the ordinary wards. Any discomfort that may arise in getting out of bed on to a cold, polished floor can easily be obviated by a pair of woollen slippers. The bathroom and lavatories should open from the sick ward and be thoroughly warmed, so that patients requiring cleansing can be easily transferred from bed to bath, and not incur the risk of chill and a possible pneumonia in their transit along a cold corridor to a perhaps colder bathroom. In the infirmary, again, it would be advisable to assign to each bed the same standard of cubic feet of air, square feet of floor and lineal feet of wall space as is adopted in isolation hospitals. This, with a less liberal allowance, should also apply to the dormitories occupied by dirty patients. Before leaving the subject of hospital equipment, I should like to point out another advantage possessed by a waxed pitch pine floor. I allude to the perpetual washings—and often with not very clean cloths—which an unwaxed floor is constantly undergoing, and which is bound to give rise to an unwholesome dampness, and may, along with overcrowding, assist not only in the production of dysentery, but, in those predisposed, of phthisis.

Treatment of the Disease.

I would preface the remarks I have to make under this head by again urging the importance, when dysentery is

prevalent among the feeble insane, of always putting the patient at once to bed on the least indication of diarrhœa, if for no other than a precautionary measure. We thus get the disease *ab initio*, and can begin energetic treatment at once. In the treatment of this, as of other affections, and notably phthisis, the remedies employed are legion; and just as various are the assertions of confidence in this or that particular one. Having regard to the different conditions under which the disease may develop, the different types it may assume, and the character impressed upon it by environment and conditions inherent in the individual, the multiplicity of drugs and various methods of treatment employed may, in great measure, be explained. Before dealing with the methods in common practice here, I should like to glance at some of the modes of treatment as employed and recommended in books by those of experience. These may be divided into that by bleeding, general and local; that by purgation; and that by emetics. From the drugs employed in the last two methods of treatment, I wish to extract two, ipecacuanha and mercury, and add a third, quinine, and place them in a separate class. Their employment I shall term treatment by means of specifics. Discussing them later on, let us treat of the other methods *seriatim*.

Venesection, etc.

Venesection, which, owing to the theories of Broussais, had been used more or less indiscriminately for all diseases, was even up to the middle of this century employed in the treatment of dysentery. In the records of the epidemics of the first quarter of the century, we find it described as "the least equivocal in its effects, and the most uniformly useful" method of treatment. To discuss the reasons for its abandonment would serve no useful purpose here. It is only one of many such anomalies abounding in medicine of the accepted modes of treatment of yesterday, finding few, if any, advocates to-day. General bleeding might be permissible, and even quite legitimate practice in acute sthenic dysentery in healthy, robust individuals, but

otherwise it is not to be thought of; although, when in vogue, it seems to have been as generally and successfully employed in advanced stages of the disease, when the patient must, of necessity, have been much exhausted.

Local bleeding, by cupping and leeching, was also employed, but why specially along the tract of the colon and in the perineum I fail to see!

Purgatives from the vegetable kingdom and salines, either with the idea of removing imaginary scybalæ or of substituting one inflammation for another, were extensively used, preference being usually given to the neutral salts, though personally I prefer the acid tartrate of potash.

Purgatives.

The varying results obtained by different observers in the employment of this drug appears to have been due to its administration in cases totally unsuited for exhibition, either from complication with other diseases, from the type assumed by the disease itself, or from climatic conditions and surroundings favouring the production of chills and thereby increasing the toxic effect of the remedy. Hence we find it extolled by some and receiving condemnation, or only a very qualified approval, from others. It seems to act most beneficially in the dysentery of tropical climates, and Hare, in an article on tropical fever and dysentery in the *Half-Yearly Abstract of Medical Sciences*, says in his concluding remarks, "The returns of the largest and longest established dysenteric hospital in the world show that since mercury has been avoided, the mortality has been double, for many years' continuance, what it was when salivation was sought for as the first and only object of treatment, and to complete the remarkable proof of the importance of mercury, these statistics clearly show that as mercury has gradually been disused, so the mortality has been correspondingly increased." As to its being pushed to the extent of salivation, opinions differ, but in any case its action must be closely watched. Although its beneficial effects are most

Treatment by Mercury.

apparent in tropical and sub-tropical countries, yet abundant proof is also forthcoming of the healthy influence it exerts on the disease in temperate climates. Calomel is the preparation mostly employed, and to it I give preference, though in some cases the liquor hydrarg. perchlor. every three or four hours seems to act as well. Sullivan says of calomel, "It is invaluable in epidemic dysentery; and is an admirable substitute occasionally for ipecacuanha; but we must carefully guard against salivation, which might prove dangerous to life."

Ipecacuanha.

The influence which ipecacuanha has on dysentery is so well known, and its specificity so thoroughly established, that only a passing reference need be made. Like mercury, the excellency of the results attending its administration are more evident in tropical than in temperate climates, and mentioning only its occasional failure in tropical dysentery, and the want of success attending its exhibition in the outbreaks in prisons, etc., in this country, I pass on to the consideration of the next specific remedy, quinine.

Quinine.

This drug, I am firmly convinced after repeated trials, is one of the most, if not the most efficacious remedy that can be used in the treatment of dysentery as it presents itself to us in asylum practice. In the various accounts of the epidemics of the 17th and 18th centuries we, find that among the remedies prescribed cinchona was often accounted the chief. Having observed the marked influence exerted by sulphate of quinine upon influenza during a severe outbreak in 1889, I determined to try it in the bowel affection, and did so with marked benefit. So much so, indeed, that now, unless for purposes of comparison, I seldom use any other internal remedy. Previously I had been in the habit of giving it in acute cases with high temperature in one large doze of twenty grains, but more, if my recollection serves me, as an antipyretic than as a routine method of treatment specially directed against the disease. On referring to my notes, I find that as far back as 1886 I gave it in some cases, but in small doses, and not with such persistence as now. In the few

cases in which it was given in fifteen grain doses thrice daily, or five grains every four hours, greater success is recorded than with other means of treatment, with the result of diminution of the purging and permanent cure. Dr Clouston, however, states that he found it of no benefit in an epidemic of dysentery in the Cumberland and Westmoreland Asylum in 1864.

The routine method now adopted in its administration is as follows. At the outset of the attack, in both sthenic and asthenic cases, and especially if the temperature be high, a dose of fifteen or twenty grains is at once given, followed by a ten or fifteen grain dose every four hours, reduced to five grains as the symptoms subside. For some reason or other at present forgotten, in my early experience of the disease I discontinued even the occasional use of quinine, and gave, in turn by mouth and enema, tincture of eucalyptus, oxide and nitrate of silver, sulphate of copper, castor oil and laudanum, etc., all with varying and perhaps doubtful success. Later, I again find myself using quinine, but in the form of salicylate, and another drug which hitherto I had not prescribed, salicylate of bismuth. Both of these were given in fifteen grain doses every four hours. The administration of these drugs, sulphate and salicylate of quinine and salicylate of bismuth, brings us now to the question of intestinal antiseptics, of late the subject of some interesting articles in the medical journals. Of those soluble in water, the perchloride of mercury is that which appears to act most beneficially in dysentery. The best results are obtained in afebrile or only slightly febrile cases in which the general health is not much undermined, and the purging not excessive. It is usually given in the form of the liquor, and in small and frequently repeated doses, fifteen to thirty drops every two or three hours. The principal intestinal antiseptics insoluble in water of which I have had any extensive experience are salol, beta-naphthol, calomel, salicylate of bismuth and the sulphate and salicylate of quinine. I have already indicated my preference for the sulphate of

quinine, but good results have also been obtained from calomel and occasionally salol. The latter, being less objectionable to the taste than β-naphthol, is more readily taken by patients, and is not so liable to set up gastric irritation. I have in several instances known a severe gastro-enteritis from the employment of β-naphthol be superimposed upon dysentery, and this reason, apart from the other objections, has induced me to entirely discontinue it. Salol is, moreover, soluble to a certain extent in oil of turpentine, and dissolved in this and made up with mucilage, to which a little oil of peppermint or cinnamon may be added, it forms a mixture which few patients object to take. In severe cases, however, I place most reliance upon sulphate of quinine and calomel.

The latter I have employed pretty frequently. Given in one large dose, fifteen or twenty grains, and repeated in smaller quantity for the next few days, I have seldom seen evil results follow, and often marked improvement. Sometimes it is given as a preliminary to the saline treatment, which is also frequently adopted.

The principle involved in the administration of these insoluble drugs consists in the fact that, in the stomach and upper portions of the small intestine, they are stated to pass through unchanged, and to split up lower down the canal into other substances. These products of chemical decomposition form other combinations supposed to possess greater antiseptic properties than the original. In what way do these insoluble antiseptics act upon the inflamed, ulcerated, and in many cases sloughing mucous membrane? Is it topically, or by so impregnating the intestinal contents as to render them innocuous, antiseptic in fact, and so prevent or retard the development of bacteria? Remembering the treatment of foul and sloughing ulcers in other regions of the body, consisting as it does in the direct application of antiseptics either in powder or solution, we should apply the same principles in dealing with intestinal ulcers.

Can this treatment be effectually carried out by the ad-

ministration alone of antiseptics by the mouth? Can the foul ulcers by this means be thoroughly cleansed, surrounded by an antiseptic medium, and irritating and putrid substances in the intestinal contents rendered harmless?

Though not denying in any way the antiseptic properties of salol, β-naphthol and subnitrate and salicylate of bismuth, I think their action in this respect in ulceration of the intestines has been greatly exaggerated. I cannot help thinking that many of the cases in which their excellence has been extolled are extremely mild, and the result of causes, attention to which and a restricted diet are all that is necessary. To say, as I have seen stated—*Lancet*, October 1895—that the beneficial results of the administration of two grains of calomel on two successive evenings in a case of typhoid were probably due chiefly to its antiseptic properties, shows a belief which, so far as ulcerative colitis is concerned I hope never to be called upon to exercise. Looking at the coloured plates of the bowel, there is forced upon one the conviction that the quantities which may with safety be given by the mouth, are totally inadequate to exert through the medium of the intestinal contents any local antiseptic effect on the wall of the colon. In cases of simple catarrh, such a view may satisfy, but in the disease we are at present discussing, the colitis, if simple—and this I do not for a moment entertain—is so for but a short time, and rapidly passes into the ulcerative and gangrenous form. Indeed, many things in the course of the disease impress one with the belief that portions of the mucous membrane slough and become gangrenous from almost the outset. I have frequently enough within a week seen the interior of the bowel present as bad an appearance as that shown by some of the plates. From a study of the conditions therein portrayed, we cannot but admit that the part played by antiseptics when given by the mouth must be indeed small.

I do not intend to assert that the drugs referred to are utterly useless, but experience has taught me that as anti-

septics in cases of ulcerative colitis, they are not sufficiently energetic; and as administered by the mouth their action is slow and in great measure destroyed by the intestinal contents. Alone, therefore, they cannot be depended upon. The acute cases are so rapid, and anything short of the most prompt and energetic treatment so dangerous, that much as we would like to see the results obtained experimentally with these substances on the growth and development of bacteria apply equally to their employment in disease, we are obliged, in the interests of the patients alone, to employ other and more efficacious means of treatment. Any action, therefore, which these so-called intestinal antiseptics exert must be indirectly through the blood. Here again the objection may be urged that the quantities required to render the blood antiseptic would poison the patient or produce a fatal gastro-enteritis. The beneficial effects are, in my opinion, not to be explained by the theory that the blood is rendered antiseptic, but rather that the drug or drugs act in a specific way on the nutrition of the parts. What this specific action essentially consists of we do not know, and at best can only theorise about. In what other way can we explain the action of quinine in malaria and mercury in syphilis? In the treatment of dysentery, I would claim for quinine the same distinction; and were I limited to one drug alone, I would unhesitatingly and with confidence give it the preference, and calomel the next place. Following out the principle which I stated should guide us in dealing with extensive ulceration of the intestines, I am very loth to trust entirely to remedies by the mouth, and usually employ in addition copious enemata, medicated or otherwise, by means of a long rubber tube (a stomach pump tube answers very well), thereby obtaining complete flushing of the affected surface. This operation is repeated several times daily, according to the requirements of the case. By this means you rapidly and effectually ensure complete removal of the contents of the colon, and a direct application to the abraded and ulcerated surface of an antiseptic solution

unmixed with intestinal contents, which would not be the case were the antiseptic given by the mouth. Hare, to whom I have already referred, seems to have been the one who most used and favoured this method of treatment. Speaking of it, he says, "The rectum is so sensitive, and so largely supplied with nerves, that when inflamed it immediately contracts on the injection and expels it, if more than three or four ounces be attempted; whereas, to wash out the colon thoroughly requires six or seven pints." And again, "By large injections we may wash out daily, with the most soothing applications, the excoriated intestine, removing its acrid secretions and the fermenting half-digested fæces, fomenting its tender surface meanwhile, and softly stretching the strictured parts with the gentle expansion of water, and when in the colon, thus daily cleansed and soothed, the inflammation and irritation have calmed, what numerous applications are there, which can be applied by the elastic tube to the whole diseased surface of the colon, from the cæcum to the anus, gently to constringe the overstrained vessels, and heal the ulcers! The great value of such medicines as alum, nitrate of silver, sulphate of copper, etc., in ulceration of other mucous membranes is notorious, and there can be no reason assigned why they should have less effect on the colon." Many of the articles dealing with the treatment of dysentery mention enemata, but as they are chiefly for the relief of tenesmus, they are of necessity small, and can affect locally only the mucous membrane of the rectum. Dr Clouston, in his account of the outbreak of dysentery in the Cumberland and Westmoreland Asylum, 1864-5, says of enemata given by the long flexible tube that the passage of the latter up the inflamed and irritable rectum caused intense and unbearable pain. This is certainly not our experience, pain only being caused by the coiling of the tube in the rectum, and even then, owing to the apathy and general insensitiveness of the patient, it may apparently give rise to no discomfort. Some difficulty in passing it may be met with, but a little patience and the

employment of a thicker rubber tube will usually overcome this. Before coming to the question of diet, I should like to mention the various medicaments I have employed in this connection, and a few other points in the treatment which I think of importance.

Foremost in the list is salol dissolved in turpentine 3i.-ii. of each to the pint of warm water. This, in my opinion, is an excellent local application, and I have never seen any ill effects attend or follow its use. Weak solutions of Condy's fluid, Friars' balsam one ounce to the pint of water, sulphates of iron and copper all have had plentiful trial. The metallic salts are perhaps to be preferred in the chronic cases, as in addition to their disinfecting properties they appear to have a more directly stimulating effect on the wall of the gut. What is essentially aimed at is in fact the interrupted irrigation of an extensively inflamed, ulcerated, and perhaps gangrenous surface by antiseptic solutions. I may add, however, that frequently warm water alone has been used. The injection should be made slowly, and no fear of rupture—at least early in the case—need be entertained. If, notwithstanding all treatment, there are no signs of improvement, it would, however, perhaps be as well to discontinue them in some cases in view of this possible contingency. Difficulty is at times experienced in passing the long tube. This is more often the case in women than in men. The knee-elbow, lateral and dorsal postures, the latter with the pelvis well raised above the plane of the head and shoulders, may all be tried; and the employment of a speculum is often of use where the sphincter muscles are sensitive, and contract on the tube, or when the patient voluntarily bears down. If the patient insists on going to a night-chair—though preferably he should use a bedpan—he should be provided with a pair of stockings and dressing gown or blanket, to prevent risk of chill.

In all asthenic and apyrexial cases, the bed should be heated by warm water bottles. When pain is much complained of, which, as already stated, is not so frequent as

might be expected, a large linseed and mustard poultice to the abdomen is often very soothing. The diet from the first, and especially in asthenic cases, must be of the most nourishing kind, in which also stimulants are indicated from the outset. Milk and beef tea or mutton soup are the staple articles, and in addition flour porridge seasoned with cinnamon is as a routine given twice or thrice daily. If the patient has previously been on boiled bread and milk for any length of time, or there is any reason to suspect a scorbutic taint, there need be no hesitation is giving lime juice or stewed fruit. The former is sweetened and a little rum added.

Diet.

ILLUSTRATIVE CASES.

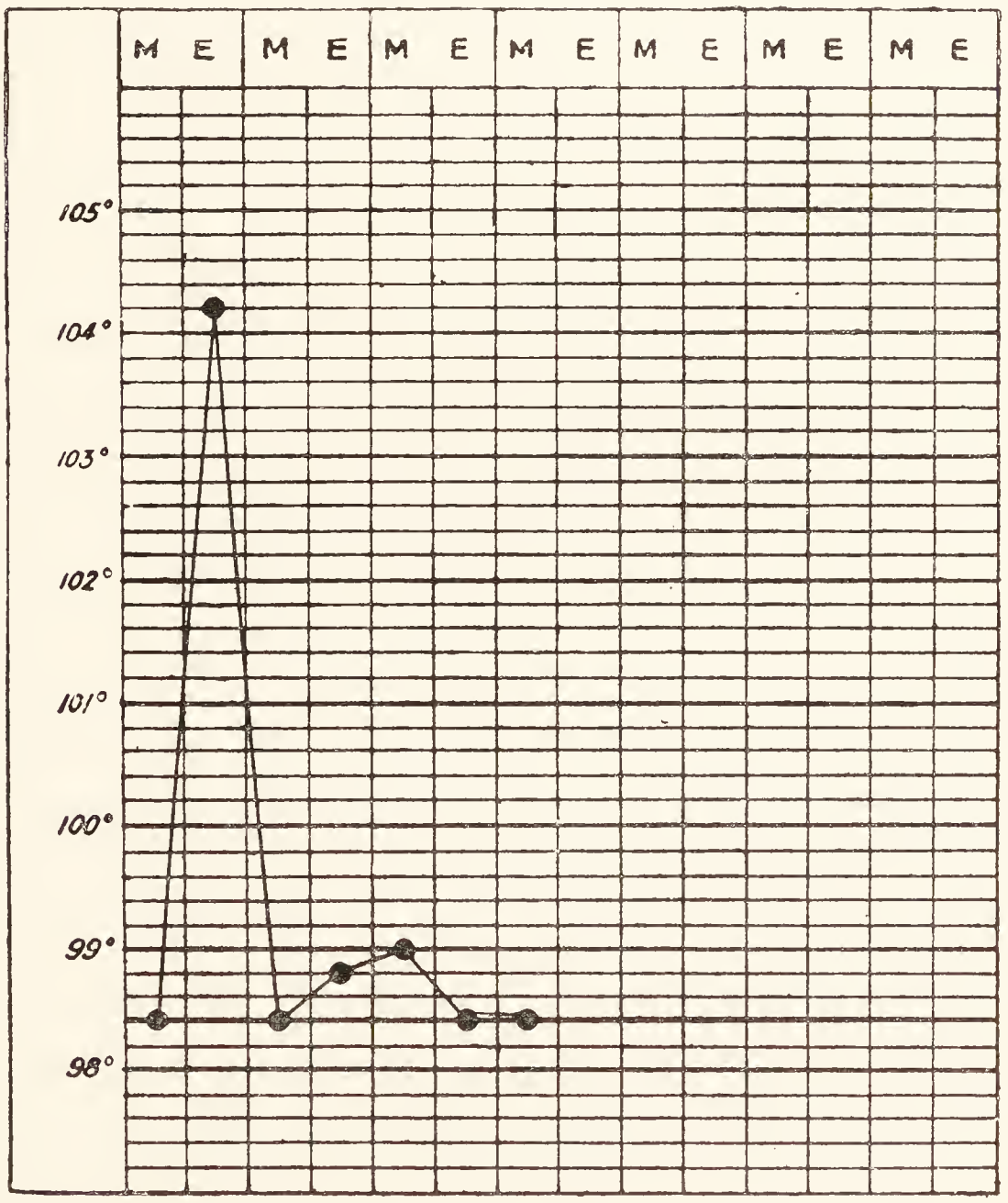
Acute Sthenic Type.

Case I. No. 54 in the *post-mortem* table.

Wm. M——, aged 27, a melancholic in good bodily health, and resident in the asylum $4\frac{1}{2}$ years, was, early in the morning on Feb. 7/95, severely purged, the evacuations being very liquid and of a greenish colour. On the afternoon of the same day, although he made no complaint, he appeared pinched and collapsed, and the temperature, which was then 103·2° F., rose in the evening to 105·2° F. There was no distension, the abdomen being flaccid, and pressure evoked no indications of pain. During the course of the day the purging continued, but the number of motions was not recorded. Treatment, quin. sulph. grs. xv. and an enema of warm linseed tea with ℥ss. tr. opii. Feb. 8. The effect of the quinine was to reduce the temperature to 103·2° F., and this morning at 8 A.M. it was practically the same, 103° F. During the night he was purged five times, the evacuations being as already described, but now containing blood. At 10 A.M. he was obviously dying, face pinched, ears and facial prominences of a livid hue, pulse imperceptible, respirations 64, and tongue dry. Stimulants freely given, but death occurred

shortly afterwards. Two points in connection with the history of this case may be mentioned; he is said to have had at the age of 13 a severe attack of intermittent fever, and at the time of the bowel complaint the weather was extremely cold, there being many degrees of frost, and the ground being covered with snow; moreover, he occupied a single room which was not heated.

Case II. Ann J. C——, aged 38, a dement in fair general



health, and resident here about 3 years. On Feb. 25/95, during the night, was purged three times, but owing to insufficient light, whether the stools contained blood and mucus could not be determined. Morning temperature normal but at

5·30 P.M. it was found to be 104·2° F. Tongue clean and dry abdomen flaccid and not painful. Urine contained no albumen. Treatment, quin. sulph. grs. xx., repeated in the evening and followed later on by a similar dose as the temperature did not fall. Long tube passed and the colon flushed out with about five pints of warm water. Bowels moved three times in the course of the day.

Feb. 26. Patient was purged twice yesterday and three times early this morning, dejecta containing blood and mucus. Treatment the same, quinine and flushing the bowel with warm water through the long tube. Feb. 27. Purged three times during the night, but as patient's general condition appeared so favourable, and she said she felt better, all treatment was suspended and recovery was rapid.

Case III. Jas. W. S——, aged 28, an epileptic dement in good bodily health, and resident 8 months.

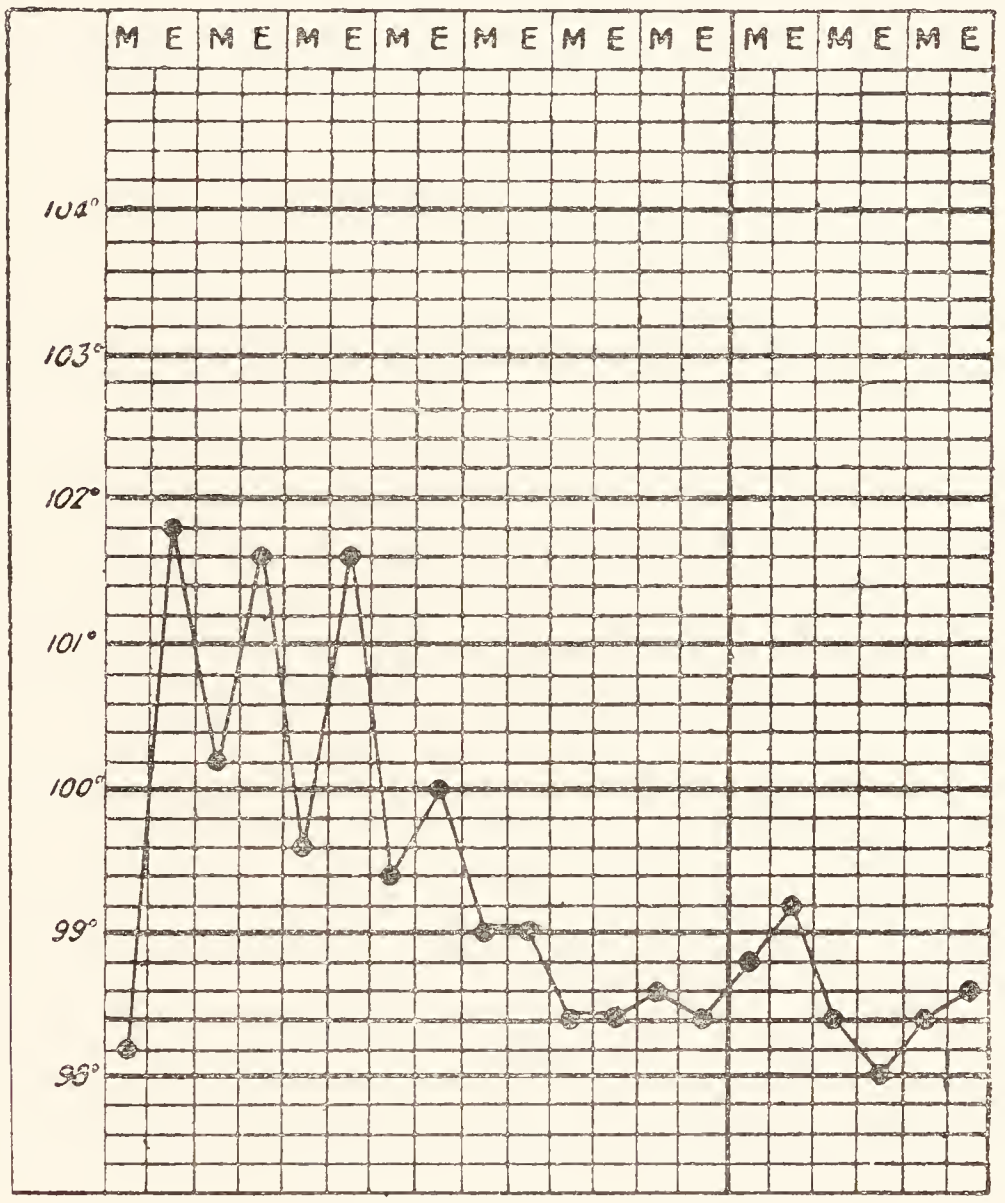
July 27/96. Was sent to bed on account of muco-sanguinolent purging and slight pain in the abdomen. The motions were scanty, and before 8 P.M. the bowels had been moved five times. Temperature, which at first was normal, rose in the evening to 101·8° F. Tongue clean, skin moist, urine normal. Ordered quin. sulph. grs. x. every four hours, milk and flour porridge. July 30. General health remains good and patient complains of little or no pain. Purging still continues, evacuations scanty and consisting chiefly of blood and mucus. On the 28th and 29th he was purged twelve and eleven times respectively, and the evening temperature on these days has been over 101° F. Quinine still continued, and to-day flushing the colon with a solution of salol and turpentine was begun.

Aug. 1. Bowel has been flushed out twice daily, and the purging has somewhat diminished, the motions numbering seven and six on the 30th and 31st respectively, and yesterday contained no blood or mucus.

Aug. 3. Purging decidedly diminishing, four times in the last twenty-four hours, and feculent. Treatment the same.

Aug. 5. Formed stool to-day, and only two motions the last twenty-four hours, feculent and free from blood and mucus. Enemata discontinued, but quinine still given during the day.

Aug. 9. Patient allowed up to-day, and may now be considered recovered.

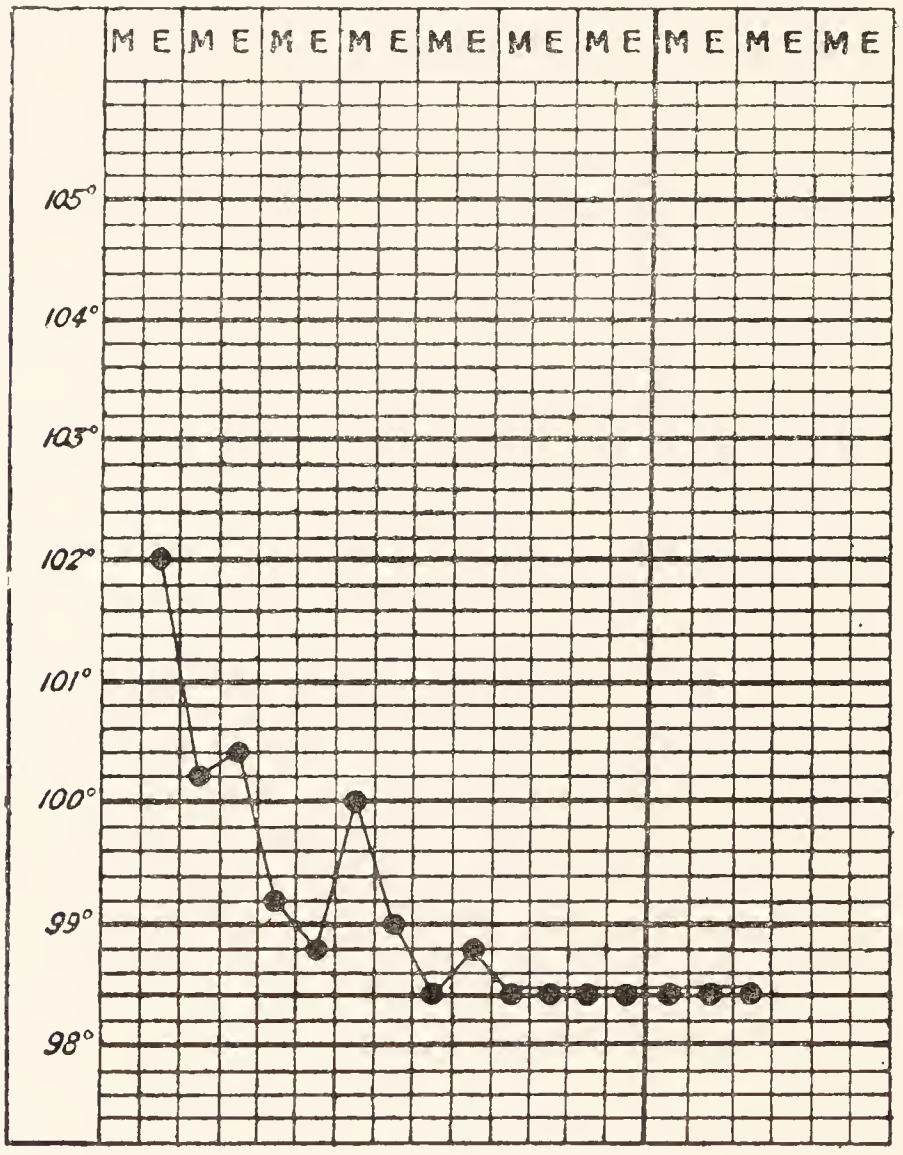


Case IV. Annie H——, aged 44, a melancholic in fair bodily health, but thin and spare, resident here 6 years.

On Dec. 13/94, was purged during the night, and temperature on being taken was found to be 102° F. This morning it is only 100·2° F. Ordered quin. sulph. grs. x. every four hours, milk and flour porridge. Dec. 15. Purging still continues ; the evacuations are thin and feculent, and yesterday contained blood. She complains of no pain, the

abdomen is flaccid, tongue clean and dry, urine normal, and this morning the temperature is normal. In the afternoon patient was sick, and vomited after the midday meal. Quinine continued as before, and the bowel flushed through the long tube with a solution of salol and turpentine.

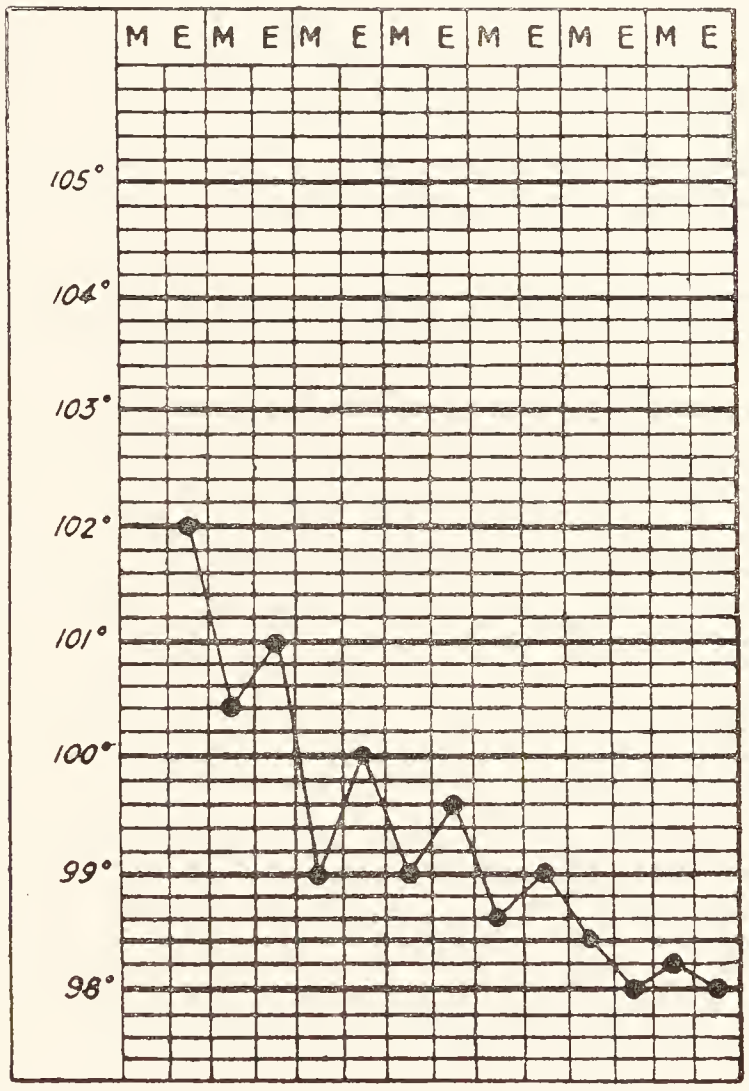
Dec. 18. Purging much diminished, there being only one motion yesterday, and to-day a slightly formed one.



Quinine reduced to grs. v. every four hours, and the enemata discontinued. Dec. 22. A formed motion to-day, and temperature for the past week has been practically normal. Dec. 26. No further purging, the motions, though rather soft, being formed. Dec. 30. Allowed up to-day, and liq. fer. pernit. substituted for the quinine. She made a good recovery.

Case V. Alice F——, aged 43, a chronic maniac in good bodily health, and resident here 3 years.

Aug. 29/95. To-day was purged three times, and temperature in the evening was 102° F. Aug. 30. During the night was purged six times, the dejecta scanty and consisting chiefly of bloodstained mucus. Face flushed, tongue dry and slightly furred, morning temperature 100·4° F., and urine normal. She complains of pain in the abdomen, increased on



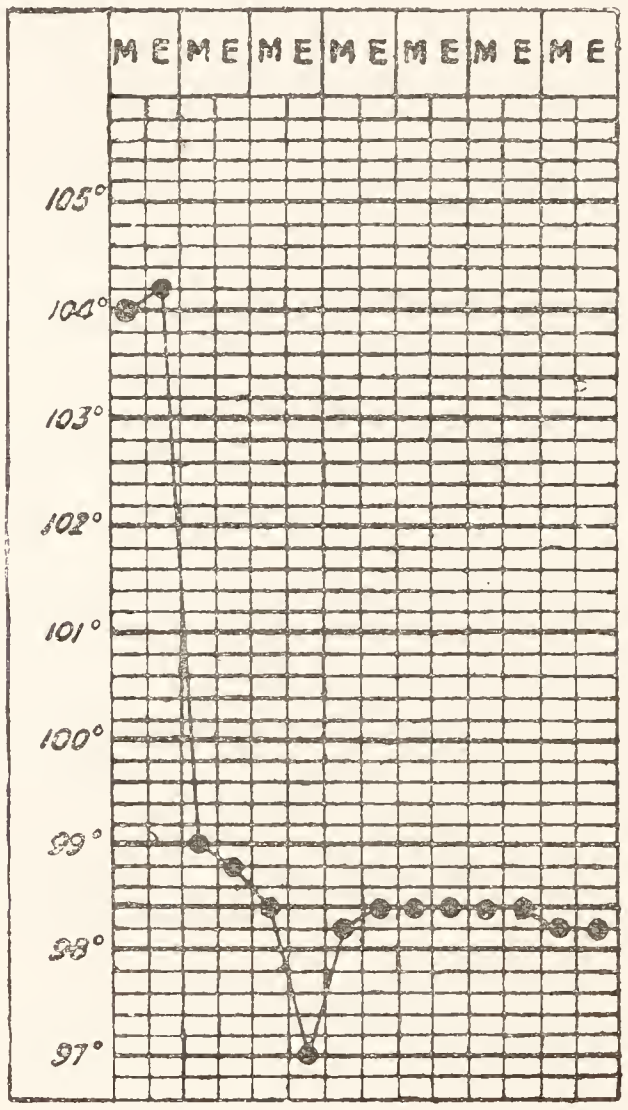
pressure. Treatment, bowel to be flushed daily with a solution of Friars' balsam, ℥i. to the pint of warm water, and patient to have every forenoon ℥ss. cream of tartar in some tepid water. Diet, milk and flour porridge.

Sept. 2. To-day the saline was alone given, and the enemata discontinued, the purging being lessened, and the dejecta showing signs of improvement. Evening temperature 99·6° F. Diet as before. Sept. 6. Two days ago there was

a formed motion, after which the bowel was flushed through the long tube. Since then all medicinal treatment has been stopped. There has been no further purging, and the patient made a good recovery.

Case VI. Geo. L——, aged 44, a dement in good bodily health, and resident 3 years.

Oct. 6/95. Yesterday was seized with a rigor, followed



shortly by a temperature of 104° F., which remained practically the same in the evening. During the night he was purged three times, the evacuations being feculent, but containing blood and mucus. Tongue dry and slightly furred. Treatment, quin. sulph. grs. x., to be repeated in the course of a few hours, salol and naphthol, grs. xv. of each every four hours, and a laxative of magnes. sulph.

Oct. 8. Purging rather excessive yesterday, the bowels

moving fifteen times. There is considerable tenesmus, and the evacuations still contain blood and mucus. Temperature normal since the second day. Treatment the same. Oct. 13. For past few days the purging has been lessened, and the dejecta free from blood and mucus. The saline is not now given daily, otherwise treatment remains the same. Oct. 16. Since last note there has been no movement of the bowels, but to-day patient was purged three times; no blood or mucus in the stools. Treatment the same. Oct. 19. Temperature last night rose to 100·2° F., but this morning is normal. All medicinal treatment discontinued to-day. Oct. 21. No further purging, and patient may now be considered well advanced in convalescence. The diet throughout the case has consisted entirely of milk.

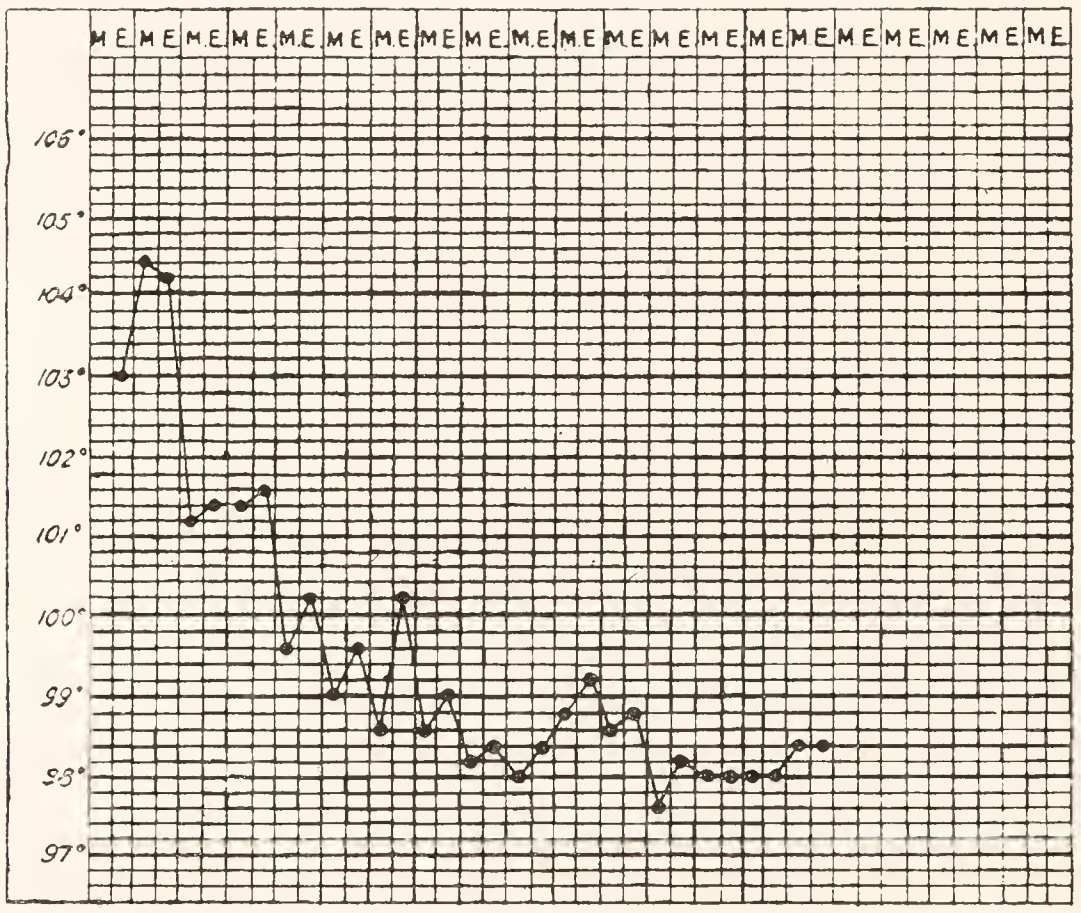
Case VII. Margt. J. B——, aged 50, a chronic maniac in fair bodily health, and resident 4 years.

Ap. 2/96. Patient took no food yesterday, and complained of vomiting and being purged. There was slight abdominal tenderness on palpation, and the evening temperature was 103° F. Tongue clean and moist, urine normal. Ap. 3. Purged eight times during the night, the evacuations being thin and feculent, and containing no blood or mucus. Face flushed, skin hot and dry, and the trunk the seat of a slight purpuric eruption. Morning temp. registers 104·4° F., evening 104·2° F. She complains of pain in the abdomen, and feeling sore all over. Purging continuous throughout the day, the stools being scanty, of a greenish colour, and peculiarly offensive. Treatment, quin. sulph. grs. x. every four hours, the bowel to be flushed twice daily with salol and turpentine solution, and diet restricted to milk and flour porridge.

Ap. 4. Purging undiminished, but temp. has fallen this morning to 101·2° F. Treatment as before. Ap. 6. Purging continues unabated, bowels moving from fourteen to eighteen times in the twenty-four hours. The stools are scanty and consist now chiefly of blood and mucus. There is no strangury or tenesmus, but complaint is made of scalding, evidently caused by

the acrid discharges from the bowel. The temp. since last note has remained morning and evening slightly over 101° F., and this morning it is 99·6° F. Treatment as before.

Ap. 9. No improvement, patient becoming very feeble and losing flesh daily. She passes everything in bed, not from any want of control, but from unwillingness to be disturbed. Temp. normal to-day for the first time. She complained of the effects of the quinine, and ten grs. of salol, along with ten drops of oil of turpentine, were given instead. Stimulants are



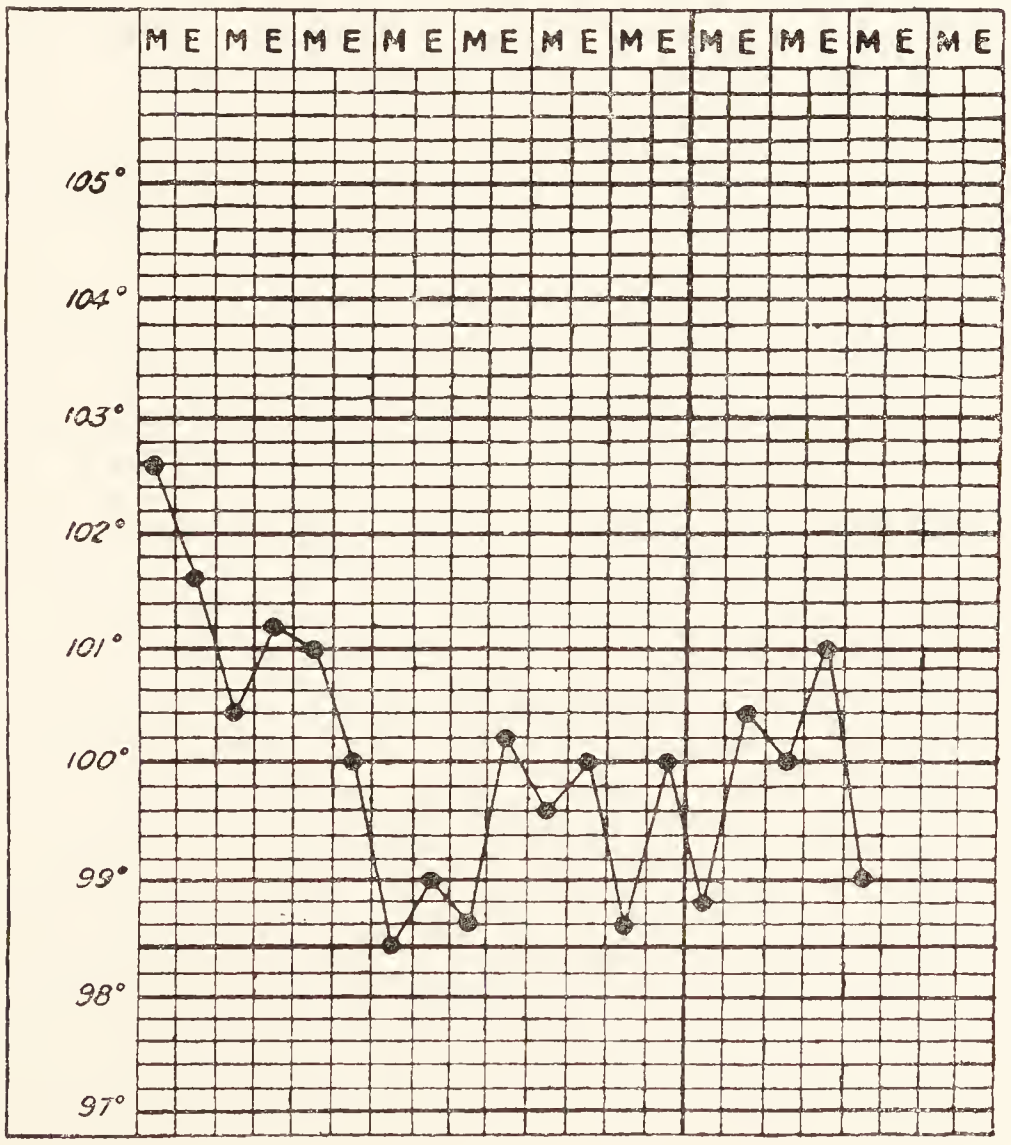
now being freely given, and the bowel is still flushed twice daily. Ap. 12. Purging has slightly diminished in frequency, but the stools still consist chiefly of blood and mucus. Temp. is now practically normal, and the purpuric rush is beginning to fade. Her general condition is very feeble, and she is fretful and irritable and unwilling to be disturbed, even when necessary. Ap. 17. General condition somewhat improved, and patient has gained in strength. Temp. continues normal, and purging is considerably lessened—five times in the last twenty-

four hours. The stools are feculent, and now contain no blood or mucus. Enemata discontinued to-day, but salol and turpentine still given by the mouth. Ap. 20. Since last note only four stools, and two of them slightly formed. General condition more hopeful, and patient is not so languid and indifferent. Treatment as before. Ap. 24. During the past three days there has been no movement of the bowels, the last purging being on the evening of the 21st. Salol and turpentine discontinued, but diet and stimulants as before.

After this the patient made a gradual progress towards recovery which it is unnecessary to describe.

Acute Asthenic Type.

Case VIII. Susan C ——, aged 75, a senile maniac, resident 3 years.



Aug. 2/95. Yesterday, patient, a fairly vigorous woman for her age, complained of feeling cold and shivery—"starved," and had an attack of bilious vomiting, followed later by scanty mucous and bloody purging and pain in the abdomen. Tongue red and dry, and temp., which at the time she was sent to bed was 102·6° F., fell to 101·6° F. in the evening. Treatment, milk, beef soup and stimulants and half an ounce cream of tartar daily. Aug. 4. Purging still continues, but to a lesser extent, and the evacuations do not contain so much blood and mucus as at first. The temp. has never been high, the evening registers since last note being 101·2° F. and 100° F. respectively. This morning it is normal. Treatment the same.

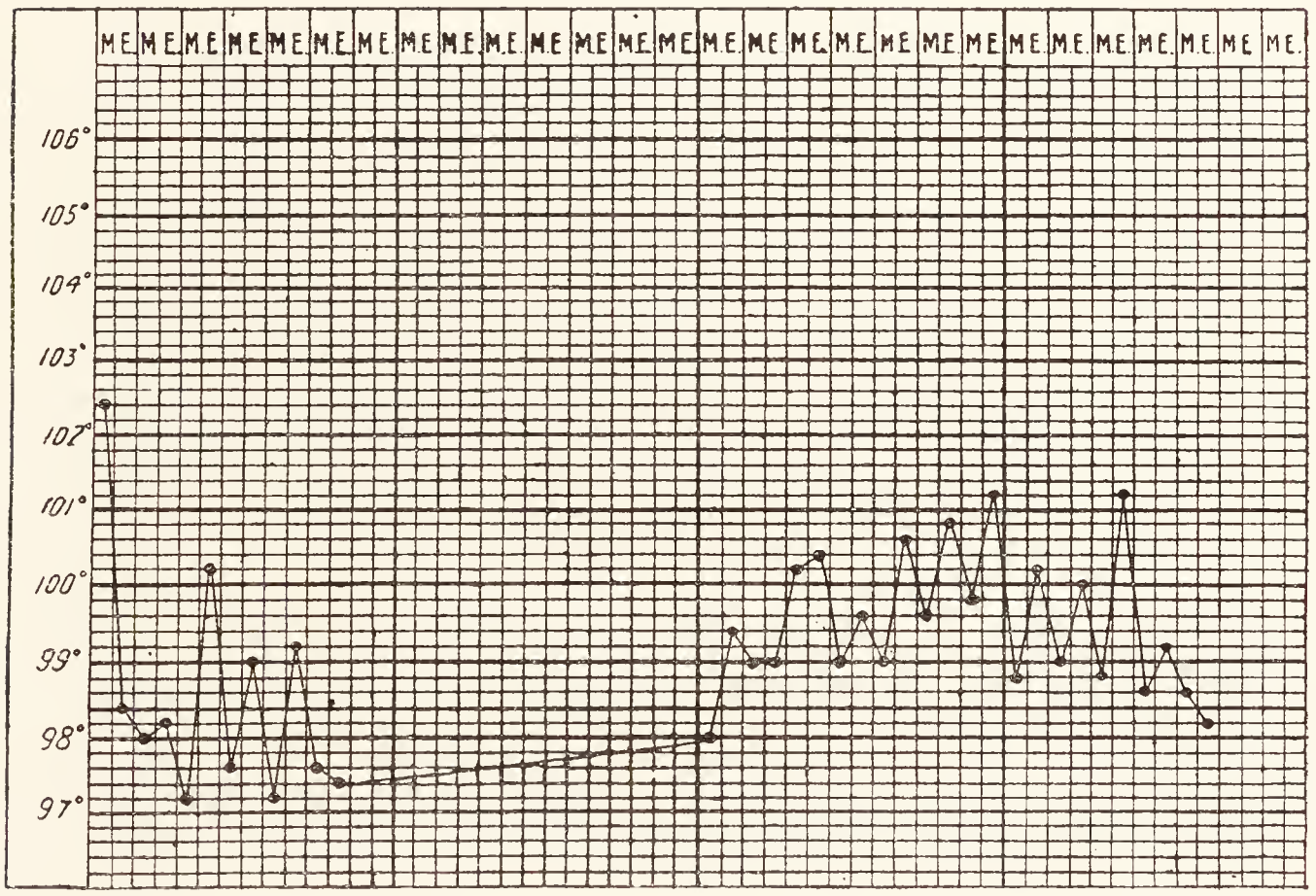
Aug. 6. Diarrhœa not so severe, but patient's general condition is grave; voice almost inaudible, hollow cheeks, sunken eyes, feeble pulse, and coldness of the extremities. Aug. 8. Bowels move about four times in the twenty-four hours, the evacuations being scanty and very offensive, Herpetic eruption on the lips, tongue dry and brown, morning temp. normal. Saline discontinued, but stimulants freely exhibited. Aug. 10. Temp., which last night was 101° F., fell this morning to 99° F. Patient has been purged six times since yesterday morning, and is sinking fast. She died in the course of the day. For *post-mortem* appearances see No. 68 in the table.

Case IX. John N——, aged 71, a senile dement in feeble health, and resident 26 years.

Ap. 3/95. About a fortnight ago patient had an attack of bilious vomiting, with a temp. of 102·4° F., which, however, fell in the evening to normal, where it practically remained during the six days he was confined to bed in the hospital. On the day following the vomiting he was purged three times, the motions being feculent and containing peas, apparently insufficiently boiled, of which the patient had plentifully partaken the previous day, and which were also present in the vomited matter. The attack was attributed to an indigestible meal, and yielded to gr. x. doses of salicylate of bismuth and flushing the bowel with warm water. About a week after

his return to the ordinary wards he was again purged and was at once removed to the hospital. In the chart this interval is indicated by a straight line.

Ap. 5. Purging very severe, as often as thirteen times in the twenty-four hours; the dejecta are muco-sanguinolent and have a horrible odour. Temp. this morning 100·2° F. Treatment, stimulants and milk diet, flushing the bowel twice daily with Friars' balsam and salol in warm water, changed, however, to-day for a solution of sulphate of iron. Ap. 10. No improvement,



the purging, with the exception of one day when there was no movement, continuing as profuse and fetid as ever. Temp. last night was 101·2° F., the highest it has yet reached. General condition very grave, features shrunken and pinched, extremities and prominences cold. Ap. 13. Temp. last night was again 101·2° F., purging unabated, stools horribly fetid, grumous in appearance and scanty. Case utterly hopeless. Ap. 14. Died to-day ; purging continued to the last. For *post-mortem* appearances see No. 63 in the table.

SHOWING THE RECTUM AND SIGMOID OF CASE IX.

Case X. Ellen S——, aged 72, a senile dement, bedridden and in feeble health, resident 18 months.

Dec. 18/95. Two days ago patient complained of pain in the epigastrium, and to-day is purged, the motions feculent and containing no mucus or blood. Tongue clean and moist, and there is no pyrexia. Treatment, milk and flour porridge, salol and turpentine mixture every four hours. Dec. 22. Purging still continues, and shows no sign of abating. Salol and turpentine discontinued, and Ol. Ric. in capsule (ten drops in each) every two hours given instead. Dec. 23. Blood observed in the motions, which are scanty, feculent, and contain no mucus. Tongue clean, dry and rough. Temp. and urine normal. Dec. 27. Purging still continues, but is not excessive. Swelling with obscure fluctuation and slight redness of the overlying skin observed in the region of the left parotid. Temp. still remains normal. There is difficulty in breathing, but owing to patient's feeble condition only a rapid examination of the chest is attempted. There are abundant crepitant *râles* all over the chest, but no dulness is detected. Dec. 28. Patient sinking, breathing rapid, pulse feeble and temp. 101° F. Died towards evening.

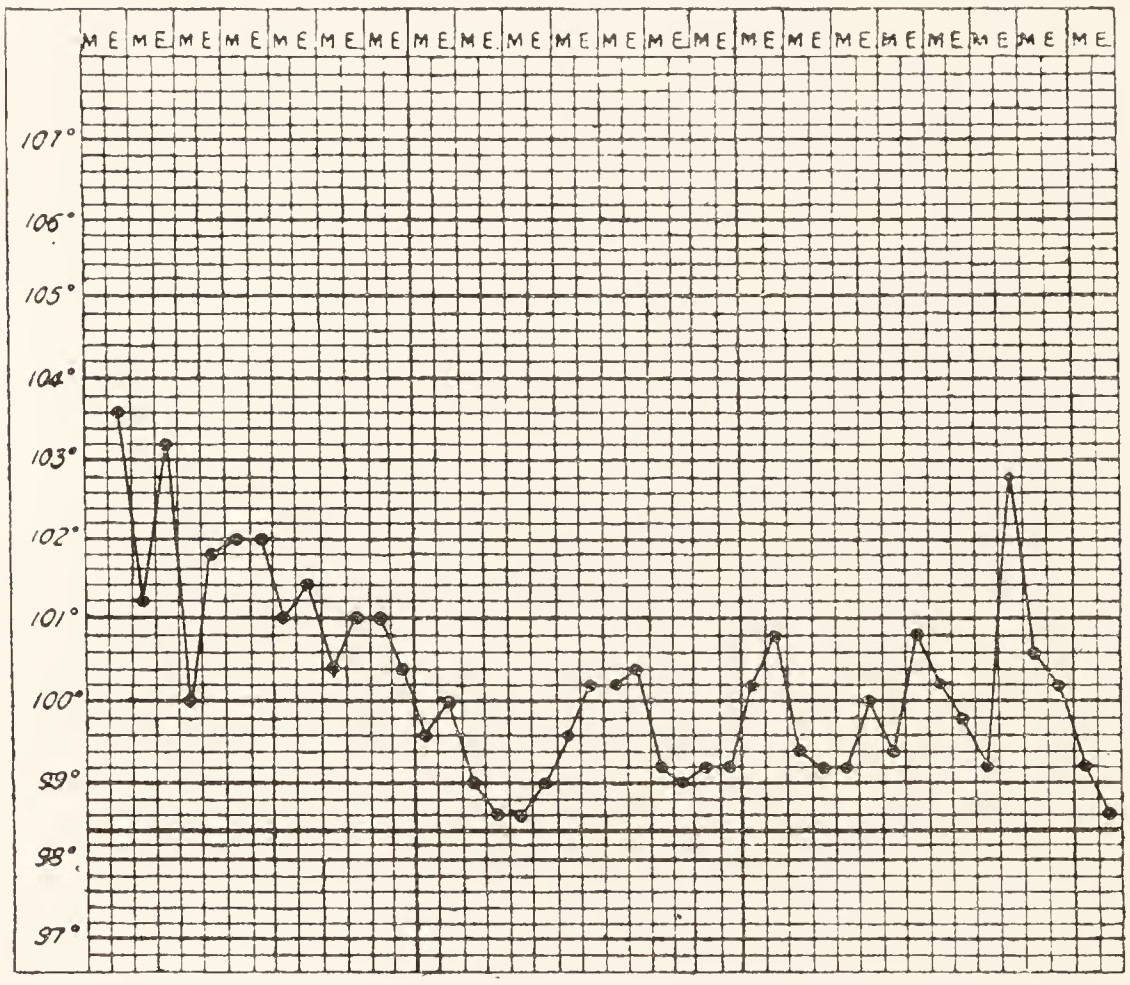
See plate I. and No. 72 in the *post-mortem* table.

Case XI. John C——, aged 72, senile mania, in feeble general health, resident 7 years.

The previous history of this patient will be disposed of as briefly as possible. He was a sailor, had frequently suffered from ague, but never from dysentery, and had been operated upon for piles some time prior to admission here. He was anæmic, very corpulent, and had a sallow, unhealthy complexion. His appetite was voracious, and he was subject to attacks of bilious vomiting, and occasionally hæmorrhage from piles, which still troubled him. On 6th March 1895 he had an attack of bilious vomiting brought on through over-eating. On the 24th he was again seized by vomiting, followed by pretty severe purging—seven times in the twenty-four hours. The stools were feculent, very offensive, and contained

no mucus or blood. Evening temp. 103·6° F., urine normal. Treatment, milk, chicken and beef jelly, stimulants, and the bowel flushed with a weak solution of perchloride of iron.

Mar. 30. No improvement, purging continues; dejecta very offensive and of a greenish yellow colour. Tongue clean and moist. Average evening temp. since last note, 101·8° F., maximum 103·2° F., minimum 100·2° F. Abdominal pain complained of. Little can be retained in the stomach owing to the vomiting. Treatment, stimulants and supportive diet



in small quantities frequently repeated, and bowel flushed once daily. Ap. 4. Patient very weak and exhausted, purging continues undiminished and the dejecta are almost entirely purulent and passed involuntarily. There is prolapsus ani and strangury, catheterism being necessary. A few days ago sulphate of iron was substituted for the perchloride in the injections, but these have now to be discontinued owing to patient's exhausted condition. Morphia suppositories give

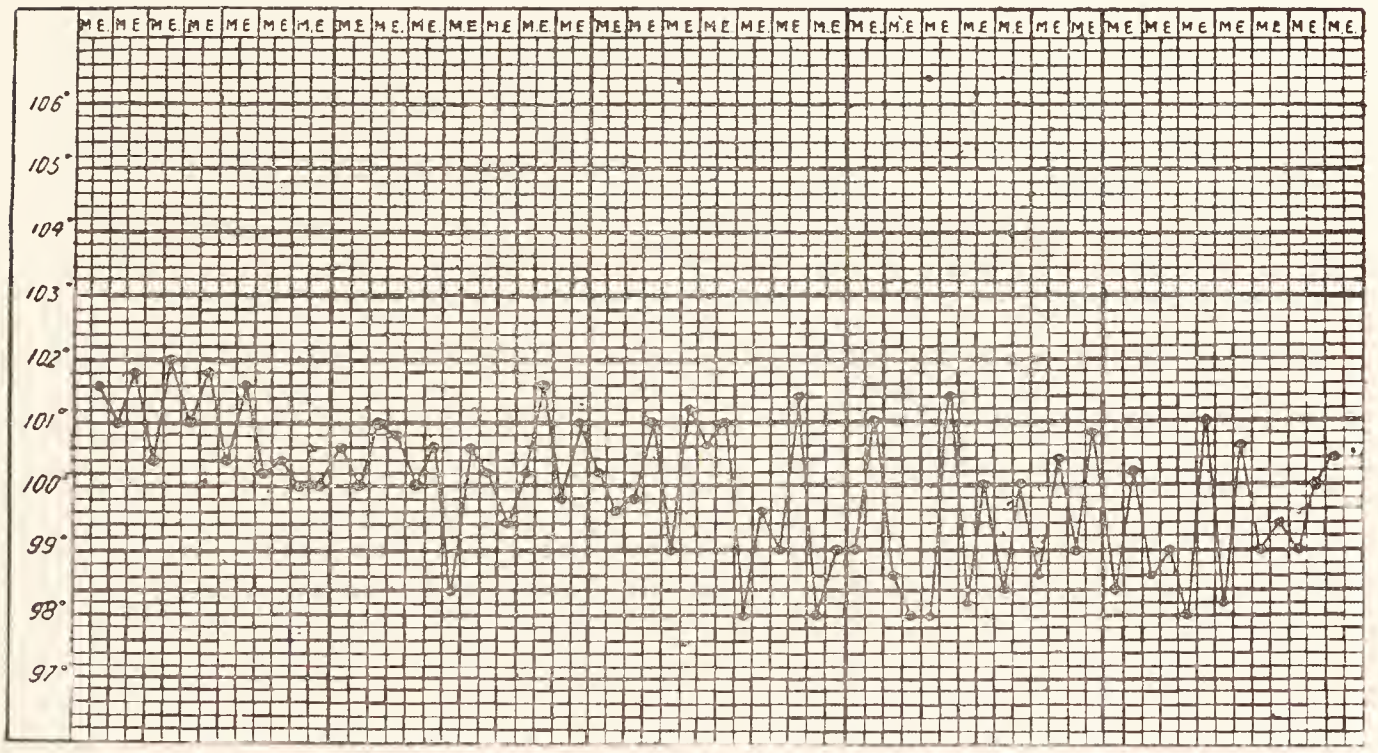
some relief to the pain, but have no influence on the diarrhœa. Temp. is lower, and since last note has never been much above 100° F., and on several occasions practically normal. Ap. 10. Condition pitiable; extreme abdominal and rectal pain, but no distension; cold, clammy sweats and feeble, fluttering pulse, involuntary purging of a puriform fluid of almost unbearable fœtor. The prolapse contributes greatly to the patient's discomfort. Even the changing of the boracic fomentations which are applied cause him exquisite pain. Ap. 13. Temp. rose last night to 102·8° F., and this morning it is 100·6° F. Patient is gradually sinking and is now quite indifferent to everything.

Ap. 14. Died this evening.

See No. 62 in *post-mortem* table.

Case XII. Wm. M——, aged 56, a chronic maniac in fair general health, and resident 7 years.

There is nothing in his previous history requiring mention



here, except the fact that early in the year, during an outbreak of influenza, he had an attack of diarrhœa, which, however, yielded to ordinary remedies and dieting.

Nov. 12/95. Sent to the hospital to-day with bloody

purging and a temp. of 101·6° F. He complains of no pain, and has no difficulty in passing his urine. Nov. 16. Yesterday the bowels moved fifteen times, and dejecta for the first time contained mucus. From the first the evening temp. has been over 101·6° F., and once 102° F.

Without describing the case in detail, I may say it ended fatally towards the end of the fifth week.

The temp. throughout was of an irregular type, with morning remissions sometimes of more than 3° F. It never rose much above 101° F., and only in the first week ever reached 102° F. Except during the first week, when both blood and mucus were present, the evacuations consisted chiefly of pale liquid feculent material of a horrible odour. The treatment adopted was salol grs. xv. every four hours, flushing the bowel with a warm solution of Condy's fluid, and stimulants. Throughout patient never showed any signs of improvement, but after the second week gradually became weaker and weaker.

See No. 71 in *post-mortem* table.

CHAPTER IV

MORBID ANATOMY AND PATHOLOGY

In the table containing the summary of the *post-mortems*, I have excluded, with one exception, all pathological changes of organs not dependent directly or indirectly upon the colitis or occurring as a complication. In other words, the lesions of organs figuring in the table have arisen during the course of the disease, and in the majority of cases are directly traceable to it. The exception to which I refer is that of the kidney. This organ is one which, next to the lungs and the bowel itself, most frequently exhibits pathological changes, and these chiefly cirrhotic. Of the cases in which this condition, varying in degree, is mentioned as being present, nine occurred in senile cases, all with one exception above 70 years of age, and whose average age amounted to 73; four occurred in general paralytics, whose average age was 41, and who had all been heavy drinkers; the remaining cases representing various types of insanity, presented ages varying from a minimum of 33 to a maximum of 65, with an average of $50\frac{1}{2}$ years. I do not mean to imply that the figures quoted in the table accurately represent all the cases of renal cirrhosis, as in many of the earlier *post-mortems* the kidney had at best been only examined with the naked eye. The systematic employment of the microscope, of course, greatly increases the percentage, and the difference it makes in statistics is very striking. It is, however, questionable how far we are justified in including in the list cases which during life show no evidence of renal

disease, and in which pathological changes in the kidney are only discoverable by the microscope. One circumstance which weighed with me in specially alluding to this pathological change in the kidneys is the importance attached to its presence by Dr Hale White in dysentery and ulcerative colitis. A reference, therefore, to the association of renal cirrhosis in this connection will not, I think, be out of place, and the present occasion appears the most suitable to introduce it. A consideration of the relationship, if any, which exists between dysentery and chronic renal disease is full of interest, and one, moreover, which has been little dwelt upon by authors. Many of the text-books and special treatises on dysentery contain only a passing allusion to the state of the urine and to the *post-mortem* appearances of the kidney. In fact, the occurrence of anyloid and Bright's disease, and the occasional presence of albumen in the urine, sums up practically all that has been written on the subject.

In the group of secondary dysenteries, reference has been made, in Chapter III. of the text, to a condition associated with chronic renal disease as productive of morbid changes chiefly confined to and most pronounced in the large intestine, indistinguishable anatomically from those of dysentery. I refer to a group of symptoms termed uræmic, dependent upon the retention in the blood of urinary constituents and characterised by coma, convulsions, more or less persistent vomiting, and frequently by diarrhœa. It is to this latter symptom that the insistence of a relationship between dysentery and Bright's disease may be attributed, more especially as has already been stated the large intestine in the latter disease is sometimes the seat of a diphtheritic inflammation. We may regard the diarrhœa in kidney disease as an effort on the part of nature to eliminate by means of the intestinal mucous membrane the retained urinary constituents. It is said to depend upon stasis and œdema, but we are also justified—according as we favour the ptomaine, or retention of urea, theories of uræmia—in looking upon it, either as the

result of a toxic poison as occurs in septicæmia, or as due to the direct application of a chemical irritant carbonate of ammonia from decomposition of urea.

What, then, are we to infer from the alleged importance of the association of renal cirrhosis with dysentery? From the point of view of prognosis, the same value attaches to the presence of pronounced cirrhosis as in any other disease, but what about it when there is no evidence of renal incompetence during life, and when morbid changes in the kidneys are scarcely or not at all apparent to the naked eye? Further, I do not feel inclined to admit that senile changes imply impaired function as regards the needs of the system. Senile pathology teaches us that certain changes are physiologically incidental to this time of life. In the viscera they essentially consist in an atrophy of the parenchyma and connective tissue. This atrophy, however, is not proportional, and consequently leads to a relative increase of the latter. I have often been struck by the similarity existing between organic tissue changes in general paralytics and epileptics, and in fact among the insane generally, indicative of old age, and those we are accustomed to meet with in the aged, and designate as senile. Atheroma, cirrhosis, fragility of bones, greying of the hair, etc., may be mentioned as the ones most frequently found. So far as the two former are concerned, after eliminating as far as possible causes likely to account for their presence, I have considered them as premature senile conditions, the functional activity and competence of the tissues affected being normal, so far as the requirements of the individual are concerned. Considering the frequency with which cirrhosis and atheroma are found *post-mortem* in lunatics who as years go may be termed young, the presence of one or other of these conditions cannot be taken, when associated with dysentery or any other disease, as anything more than accidental.

Dr H. C. Bristowe, in an interesting article in the *Journal of Mental Science* for April 1895, suggests that the great

frequency of the association of general paralysis with renal cirrhosis may be due to one and the same irritant. Without, however, entering into any discussion relative to the frequent occurrence of cirrhosis of the kidney and atheroma in the insane generally, I may be permitted in passing, to draw attention to the fact that notwithstanding, symptoms referable to the kidney and rupture of diseased blood vessels are comparatively rare, whereas among the sane in our large workhouses they are very common. A possible explanation may lie in the different origin of the cirrhosis and atheroma in the insane, the even, uneventful existence which many of them lead, and their plain and regulated diet. My reasons, therefore, for attaching only an accidental importance to the frequent association of renal cirrhosis and dysentery, or for the sake of argument simple ulcerative colitis, may be tabulated as follows:—

1. In many of the cases of dysentery the kidneys were perfectly healthy, or the cirrhosis slight and discoverable only by the microscope.

2. Many of the cases of cirrhosis were unattended during life by any evidence of disturbance of renal function, and were often associated with other degenerative changes, which, from the age of the patient and the time he had been under observation, could not be attributed to the ordinary causes of such degenerative changes, and which may be designated premature senile changes without functional incompetence. As proof to a certain extent of the renal function being intact, we may mention that, apart from the occasional detection of albuminuria, any indication of serious organic disease of the kidneys is comparatively rare.

3. In general paralysis of the insane, it is generally admitted that renal cirrhosis, from whatever cause arising, is present in a large percentage—72 per cent. according to Dr Bristowe—and yet experience here and at Whittingham Asylum of hundreds of deaths in general paralytics seems to show that, though forming a large proportion of the total

number of deaths, they by no means compose the bulk of the cases dying of dysentery.

4. If renal cirrhosis had any other than an accidental association with dysentery, how can we account for the latter not being much more common than it is in private and hospital practice? Or how explain the fact that it is more or less endemic in some asylums, others showing almost complete exemption? And again, how explain the seasonal influence on the bowel affection and the numerical differences between one year and another?

Regarding the case of thrombosis of the left renal vein, it may possibly have been due to an extension of the inflammation from the adjoining much diseased descending colon, or to the cardiac condition and extreme atheroma of the abdominal aorta, which was rough and irregular, from calcareous plates.

Returning now to the condition exhibited by the other organs, we find in the lungs pneumonia present in one or other of its forms on eleven occasions—a large percentage. Three of these were undoubtedly septic, one being also associated with metastatic abcesses in the left parotid. The mesenteric glands showed pathological changes, consisting chiefly of enlargement and congestion, in only eighteen cases, or 22 per cent. In only one instance had the change, gone on to suppuration. Considering the favourable opportunities that exist for septic absorption and inflammation, the fact that the glands are not more frequently involved may be taken as in part evidence that the poison neither originates in nor travels by the lymphatic glandular system. The occurrence of pyæmic abscesses in the liver, lungs and parotids, though not very common, are still sufficiently so to suggest the veins as the means of conveyance. Contrast this comparative freedom of the mesenteric glands in dysentery with what takes place in tubercular ulceration of the intestines, in which it is exceptional to find them unaffected.

Hale White, in his description of simple ulcerative colitis, says enlargement of the mesenteric glands is very rare.

The congestion of the stomach can be accounted for, I think, by the contiguity, and in one case the adhesion, of the inflamed and ulcerated transverse colon. In one case, No. 32 in the table, diphtheritic patches are mentioned as existing at the lower end of the œsophagus. The only other instance I can find of this part of the alimentary tract being implicated is in a case at the Whitworth Hospital, Dublin. The lesion is described as a very vascular patch to the right of the œsophagus. As the patient had been salivated and the stomach, moreover, is stated to be coated with a yellowish glairy secretion, it is possible that the condition was caused by the use of mercury.

No. 73 might, perhaps, be better designated as a case of follicular colitis. The small intestine is seldom affected by any specially pronounced morbid lesion, erosion occurring in only seven of the cases. In nineteen cases it is described as congested, and this is limited to the last foot or less of the ileum, and is caused by contact with the diseased colon. Where patchy areas of congestion occur throughout its length, I believe this is due to the same cause, such as close proximity in the pelvic cavity to an inflamed and ulcerated rectum. It is interesting to contrast the changes occurring in dysentery with those of tubercular disease of the bowel. Here, in the great majority of cases, the morbid lesions are confined to the small gut, or, at all events, are more pronounced there. Dysenteric ulceration, on the contrary, is limited to the colon, the participation of the small bowel, when it does occur, consisting chiefly of deep congestion. Further, the general appearance of the mucous lining and ulcers of the large intestine, when the seat of tubercular disease, present an appearance in no way resembling that found in dysentery. In the former disease they are—unless in the cæcum and rectum, where they are closer together and often coalesce—small, circular and have a punched out appearance, and are separated by large tracts of pale but otherwise apparently healthy mucous membrane, in which, however, on holding

it up to the light, enlarged solitary follicles may often be seen. In no case, moreover, does one see the large, irregular sloughs and ulcerations separated from each other by narrow strips of inflamed and eroded mucous membrane. One circumstance in which tubercular ulceration of the large bowel resembles dysenteric ulceration is the infrequency—as compared with its occurrence in tubercular ulceration of the small intestine—of perforation and peritonitis.

In the abridged table are included, so far as the large bowel is concerned, only cases exhibiting the most marked changes in the colon and rectum, with the object of conveying, in a tabulated form, the profound alterations to be met with. The following description, then, applies to the general appearances presented in the usual run of cases.

General Description.

The serous covering is only exceptionally altered, and inspection as a rule gives no clue to the condition existing within. On opening the bowel what strikes one, after observing the extensive destruction of the mucous lining, is the sodden and infiltrated appearance of the walls, the variety of colours and different stages of ulceration, and often a gangrenous-like odour. There is generally no special localisation of the ulceration to any particular region of the bowel to the exclusion of intermediate portions which remain healthy, although the changes are often most marked in the cæcum, flexures and rectum. The entire thickness of the mucous coat seems to be involved. In sthenic cases dying in an early stage, the interior is covered with a thick layer of mucus more or less bloodstained, easily removable by washing, and beneath which the inflamed and eroded coat is distinctly made out. Or it may exhibit over a varying extent of surface a croupous or diphtheritic exudation, using these terms as signifying a superficial or deep coagulation-necrosis, and sometimes this condition may extend continuously from

cæcum to anus. [See No. 78 in the *post-mortem* table.] The asthenic cases generally may be said to be characterised by the presence of large, unhealthy, irregular ulcers, extensive sloughs, yellow and infiltrated with pus, or gangrenous and of a dark green hue.

The erosion varies in extent and degree, and gives the interior of the bowel a rough, granular appearance and feel. It may be quite superficial, involving only the free extremities of the glandular surface, or extend in many cases to complete denudation of the mucous coat. The ulcers vary greatly in size and depth, and in severe cases, as may be gathered from what has already been said, the muscular coats are also involved in the necrotic process, and are sodden, easily lacerated and of a greyish green colour. The areas of mucous membrane between the ulcers, where these are numerous and not far removed from each other, give an irregular, trellis-work look to the whole interior, and the floors of the ulcers present a fine, fretted appearance, owing to the prominence of strands of connective tissue and the areolar tissue network of the different layers. The colour of the parts varies in different cases, and all shades, from an angry red to a livid purple and even blackish hue, interrupted by paler areas of a greyish green, or the aforementioned sloughs or membranous exudations, may be seen. In cases where the mucous and muscular coats have almost or entirely disappeared, the binding connective tissue, which is the last to vanish, and is often greatly thickened, is applied to the serous coat, and may thus assist in the prevention of mechanical rupture, perforation having been observed in only one of the eighty *post-mortems* recorded. The sloughs vary in size, and are isolated or partially surrounded by inflamed and otherwise altered mucous membrane. They are not, so far as my observation goes, often discharged in masses with the stools, but seem rather to undergo a process of disintegration within the bowel, appearing in the evacuations as small, shreddy pieces of connective tissue. In the

diphtheritic form, separated portions, representing the fibrinous exudation, along with a considerable thickness of the glandular layer, are more frequently met with than sloughs involving the entire thickness of the mucous coat, the process of separation being slower in the latter and thus giving time for a gradual disintegration. In No. 83 in the *post-mortem*, and plate No. II., these superficial fibrinous sloughs were often found in the dejecta. I have referred more than once to the sodden and infiltrated look of the walls, and I may here take the opportunity of leading into the microscopical appearances by stating that it depends upon a fluid exudation from the vessels chiefly of the submucosa.

Microscopical Appearances.

It will, perhaps, at this stage, in order to thoroughly appreciate the minute morbid anatomy, not be superfluous to briefly enumerate a few points in connection with the normal structure of the mucous and submucous coats of the large bowel. All that need be said of the former is, that it is chiefly composed of the crypts of Lieberkühn placed vertically and in close apposition, and which secrete mucus. Concerning the latter, however, there is one point to which I wish to direct special attention. I refer to the position of the solitary follicles. These, which are closely allied if not actually identical in structure and function to lymphatic glands, are here situated entirely in the submucosa. They do not, as in the small intestine, pierce the muscularis mucosæ, and, pushing aside the tubular glands, approach the lumen of the gut, from which they are only separated by a single layer of columnar cells. Their position varies, some being placed in the centre of the submucosa, others abutting against the muscularis mucosæ, which may even appear to split and embrace the follicle. They vary also greatly in size even in the same bowel, as the accompanying wood-cuts demonstrate, and are recognised as a collection of leucocytes

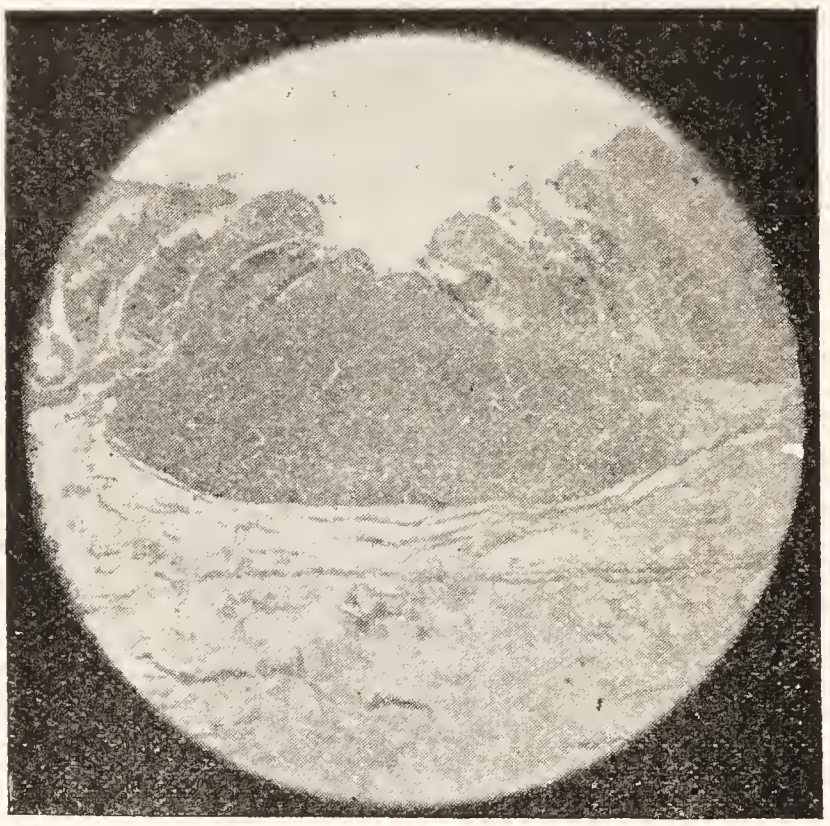


Fig. 1.

Showing variation in size of solitary follicles from normal colon.

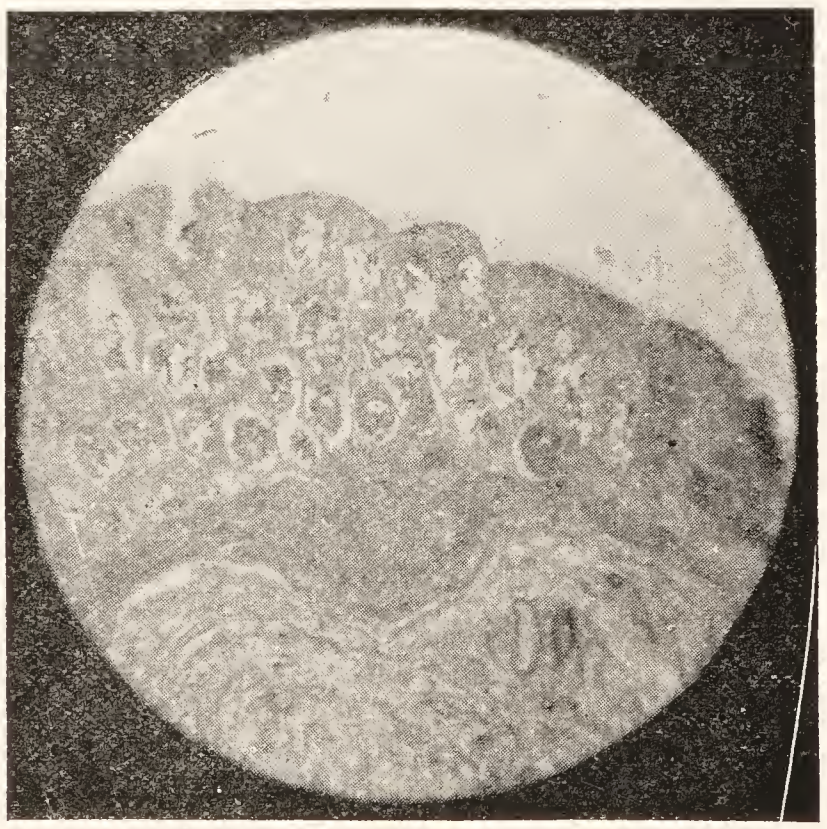


Fig. 2.

Sections showing variation in size of solitary follicles from a normal colon.

distinctly demarcated from the surrounding submucous tissue. As placed, then, in the colon, the entire thickness of the mucous coat, along with the muscularis mucosæ, intervene between them and the intestinal contents.

The submucous coat resembles that of the small intestine. It consists of loose areolar tissue, and contains the blood vessels and lymphatics, and, as already stated, the solitary follicles.

We have sufficiently indicated for our purpose the chief

Fig. 3.

Section showing variation in size of solitary follicle from a normal colon.

peculiarities of these two coats, and now let us see what changes they undergo in dysentery.

This affection has been described as a specific colitis, a specific inflammation of the mucous membrane of the large bowel, which, according to the degree of intensity manifested, has received the names of catarrhal and croupous-diphtheritic. These two latter terms, arguing from their application to inflammation of other mucous surfaces, would seem to indi-

NORMAL BOWEL.

LOW POWER.

Fig. 4. Showing mucous and submucous coats.

HIGH POWER.

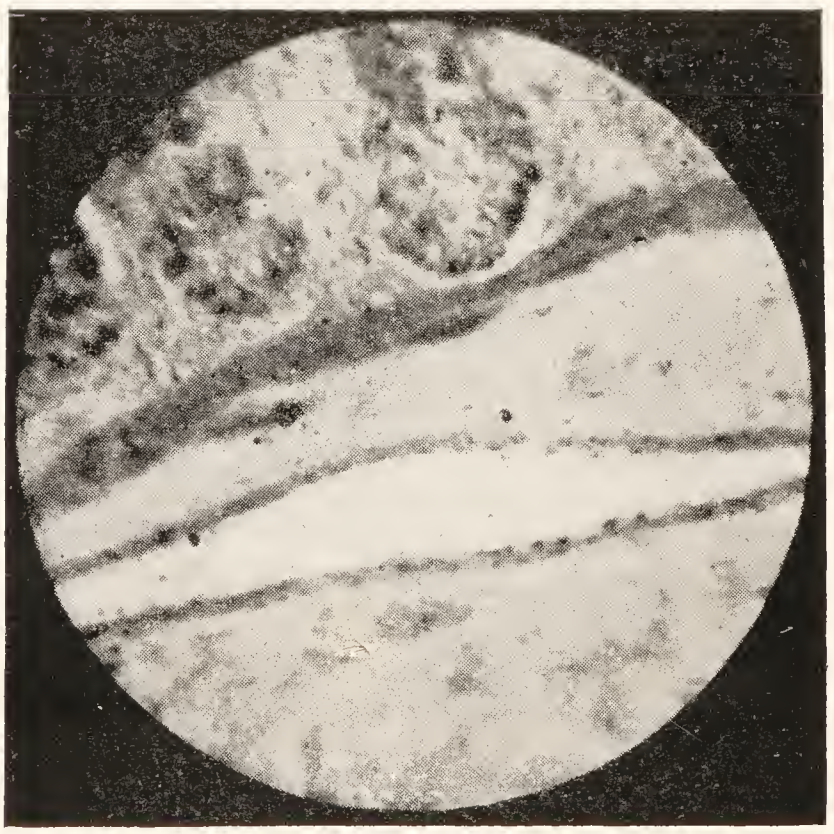


Fig. 5. Showing lymph channel.

cate that it is the mucous membrane which is primarily and principally involved. In the article on dysentery in *Quain's Dictionary of Medicine*, Ewart says, "The dysenteric process generally consists of a specific inflammation of the solitary glands." Again, "The adjoining follicles of Lieberkühn do not in these cases necessarily participate to any great extent in the diseased process." This would seem to imply that the disease begins in the solitary follicles, and that the mucous coat—the follicles, be it noted, lying in the large intestine entirely in the submucosa—is involved secondarily to the follicles, which are lymphatic in structure and function, but to which, by the way, the writer just referred to ascribes a secreting function, which, in the ordinary acceptation of the term, is as here applied misleading.

Let us take as examples of specific follicular inflammation those occurring in typhoid fever and tuberculosis. In both, the chief seat of the lesion is in Peyer's patches, and only the mucous coat in their immediate neighbourhood participates, and that secondarily, in the inflammation. In both, again, the morbid processes are not necessarily confined to the follicles of the small intestine, for those of the colon may also be involved. Owing, however, to the more scattered position which they occupy in this portion of the bowel, except in the cæcum, where they are more numerous, the inflammatory manifestations are not so extensive as in the small intestine, and give rise in many cases to what has been termed follicular ulceration. In only one instance have I witnessed anything approaching this in dysentery. The follicles were prominent, just beginning to ulcerate, and could be well made out on holding the bowel up to the light. The case occurred in a very feeble woman aged 72, and was fatal on the twelfth day. [See No. 73 in the *post-mortem* table.] Another feature of primary inflammation of lymphatic tissue, as exemplified in the two aforementioned diseases, is that it is more circumscribed in its local effects than when a surface is primarily attacked by inflam-

mation, specific or not. Contrast, for example, the extensive superficial area in erysipelas, phlegmonous erysipelas and diphtheria, with the more limited manifestations around the lymphatic glands in other infective diseases, in which the surface is attacked secondarily to the glands, as occurs in soft chancre and tubercular disease. No one surely would assert that in erysipelas and diphtheria the glands are first affected and thence outwards the skin and mucous membrane. Why, then, in dysentery should we seek to locate the disease as starting in the solitary follicles! Erysipelas and diphtheria are both acute local infective diseases of surfaces dependent upon inoculation of specific micro-organisms. They are specially liable to occur where insanitary conditions are present, and when these persist may even become endemic. They are also epidemic and contagious, and are frequently to be met with in those institutions where dysentery prevails. There seems little objection to urge, then, from these and other considerations, against placing dysentery in the same category as erysipelas and diphtheria. The term diptheritic is indeed applied to dysentery as indicative of the character of the inflammation, and the same idea seems to have been in the mind of Cullen in speaking of the enteritis, when he says, "The inflammation of the intestines may be either phlegmonic or erythematic." I am strongly of opinion, then, that the involvement of the solitary follicles in dysentery is secondary to that of the mucous coat, which is the tissue first attacked.

Let us see now what occurs in dysentery. To begin with, we have the disease limited exclusively to the large gut, the small intestine presenting, as a rule, a perfectly normal appearance. The exception, when it occurs, is in the last 10 or 12 inches of the ileum, and is due to irritation set up by contact with the inflamed and ulcerated cæcum or rectum. The morbid process, as observed in the colon, consists of an acute inflammation, with at first a superficial erosion of the mucous coat, but without any evidence of special involvement of the follicles

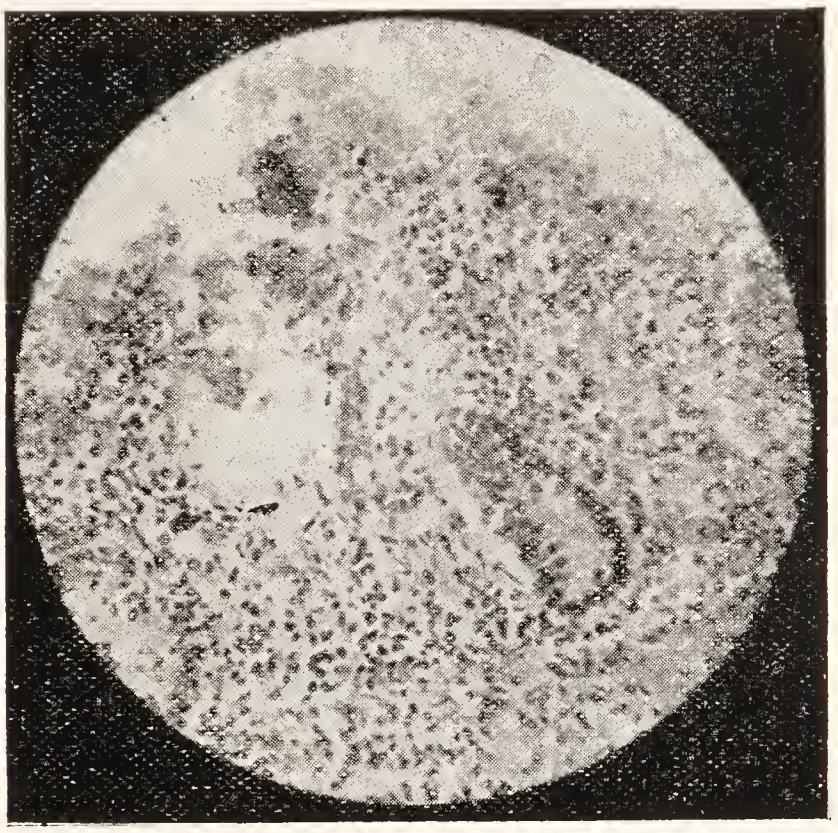


Fig. 6. Showing cell infiltration and destruction of Lieberkühn's glands.

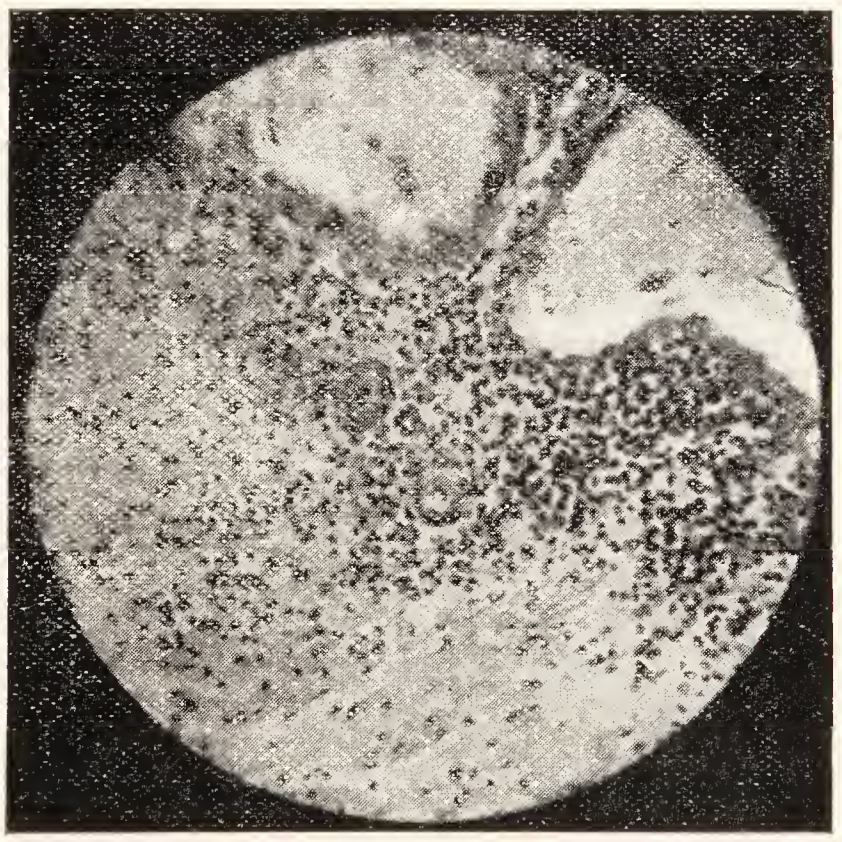


Fig. 7. Cell infiltration of submucosa, with dilated lymph spaces.

as occurs in typhoid and tubercular disease. The inflammation again, in dysentery is much more extensive than in these two diseases, involving in many cases the entire mucous surface from cæcum to rectum, with few, if any, healthy portions intervening, and similarly if only one of the divisions of the colon be affected.

A careful examination of the bowel in fatal cases of four, five and six days' duration, and a comparison with the normal mucous membrane has convinced me that in no instance are the follicles affected without the mucous coat in the first instance showing distinct evidence of destructive inflammation and cell infiltration. Figures 6 and 7, both from the same case, show this cell infiltration. In 6, a little to the right is a remnant of one of Lieberkuhn's glands, and in 7, note the distension of the lymph spaces, and the obliteration of the wavy character of the submucosa by the mass of exuded cells. The follicles, in fact, only participate in the general cell infiltration of the mucous and submucous coats, and though at first their outline is distinguishable, it is soon lost trace of in the general exudation. They become distended and rupture, and their contents mingling with the inflammatory products, all evidence of their original structure is lost. Occasionally, however, we can demonstrate them as distinct from the surrounding tissues, as shown in Figures 8 and 9, the former from a case of twelve days, the latter from one of five weeks' duration. That, however, the follicle is morbidly enlarged in either is open to doubt, as they exhibit great variability in this respect in the normal bowel (Figs. 1, 2 and 3). To locate dysentery, then, as originating in a primary inflammation of the follicles, with extension afterwards to the mucous coat, does not appear to exactly coincide with the evidence. I maintain, then, that their involvement is always secondary to that of the mucous coat. The explanation, therefore, of the sago or frog-spawn clumps of mucus in the stools being due to gelatinoid moulds of follicular cavities becomes thus purely imaginary. I have frequently observed

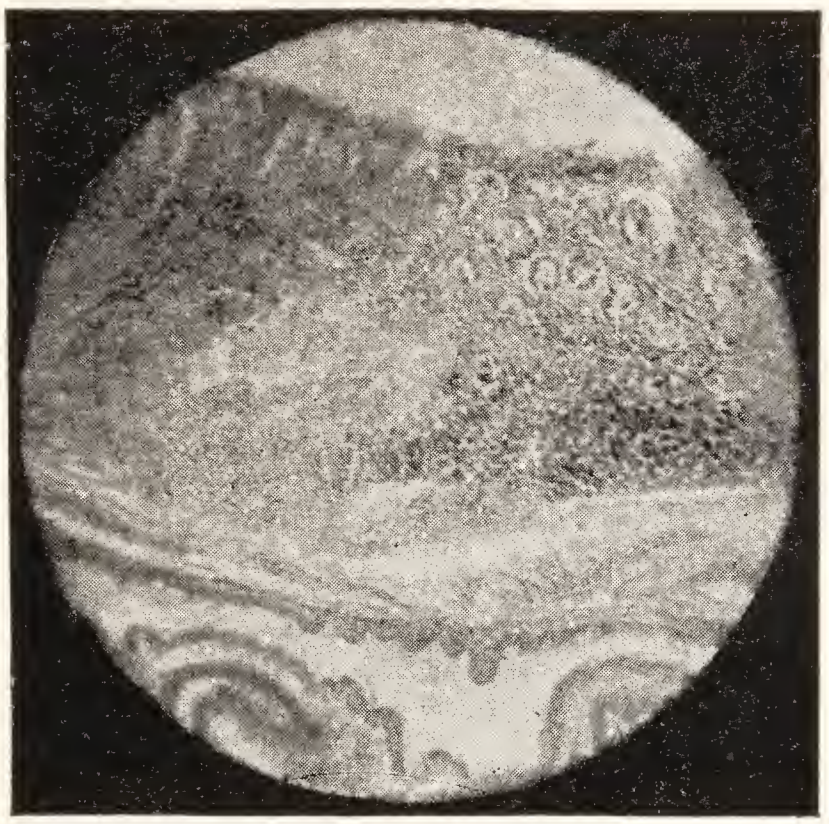


Fig. 8. Erosion and cell infiltration of mucous coat. (*a*) Follicle before it is obliterated by surrounding cell exudation.

Fig. 9. (*a*) Follicle distinct from (*b*) commencing development of fibrous tissue.

them in the evacuations and on the mucous surface *post-mortem*, and regard them as caused by rupture of occluded and distended tubular glands, or by collections of mucus in the hollow of an ulcer.

In Figures 10 and 11 are represented the appearances of the two inner coats in an early stage of the disease, before complete destruction of the mucous lining and rupture of the muscularis mucosæ. They portray a state of superficial erosion, in which the free ends of the glands are destroyed and their outline defaced by cellular and fibrinous exudation. In Figures 6 and 7 is also shown under a high power a somewhat similar condition.

In cases characterised chiefly by fibrinous exudation, the mucous coat, partially or in its entirety, is infiltrated and destroyed, according to the severity of the inflammation. This pathological condition has given rise to the terms croupous and diphtheritic as applied to dysentery, but it is to be understood that they imply only a difference of degree.

The mucous coat may exhibit only this fibrinous exudation, or with or without it be the seat of extensive sloughs and separation.

The ulcers, as already indicated, vary greatly in size. Their floor and sides are formed usually of the much altered mucosa, though sometimes this and even the muscular coat have disappeared, and only toughened connective tissue strands and thickened peritoneum remain. Their edges are fringed with irregular projections of separated mucous membrane, or the latter may gradually shade off as it approaches the ulcer with which it eventually blends.

The submucosa soon becomes altered by the development of fibrous tissue. This is best seen in sections made through healing ulcers, or in chronic cases where it may be very thick and tough.

Little more remains to be said, beyond mentioning the so-called polypoid formations. They may be formed from portions of mucous membrane which have assumed undue

Fig. 10. Showing superficial erosion with cell infiltration of mucous and submucous coats. Low power.

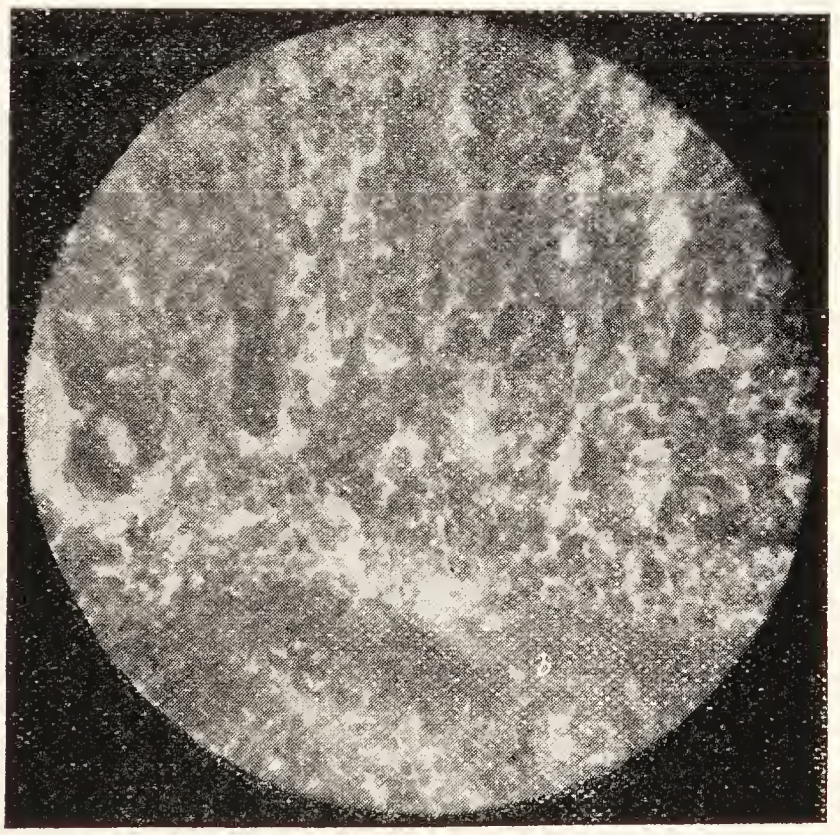


Fig. 11. Cell infiltration of (*a*) mucosa, (*b*) muscularis mucosæ, and (*c*) submucosa. High power.

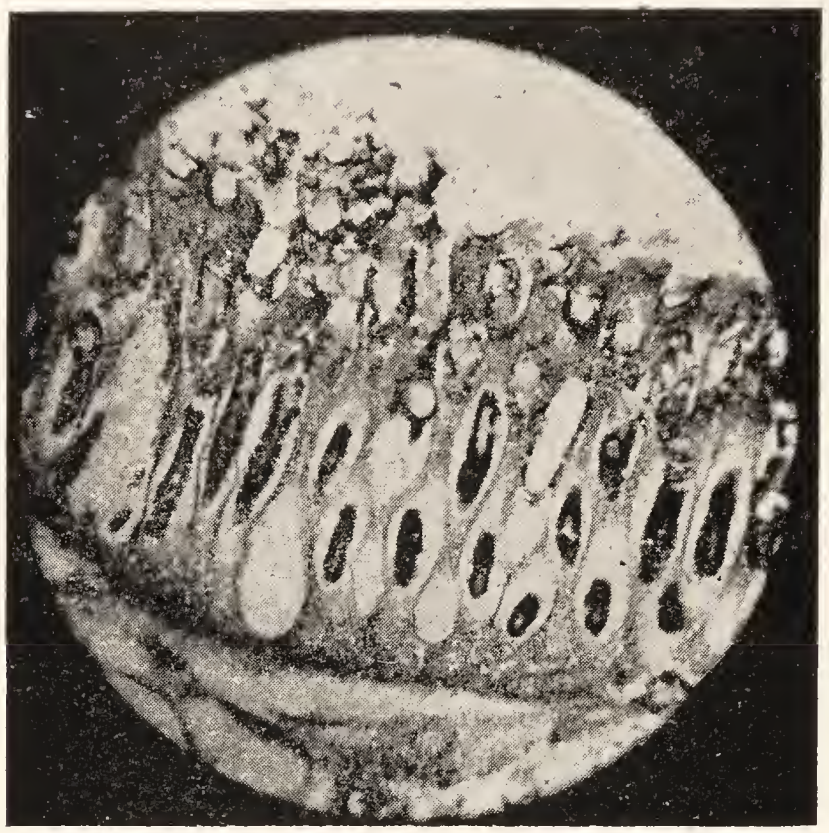


Fig. 13. Croupous dysentery, showing at (*a*) superficial destruction and fibrinous infiltration.

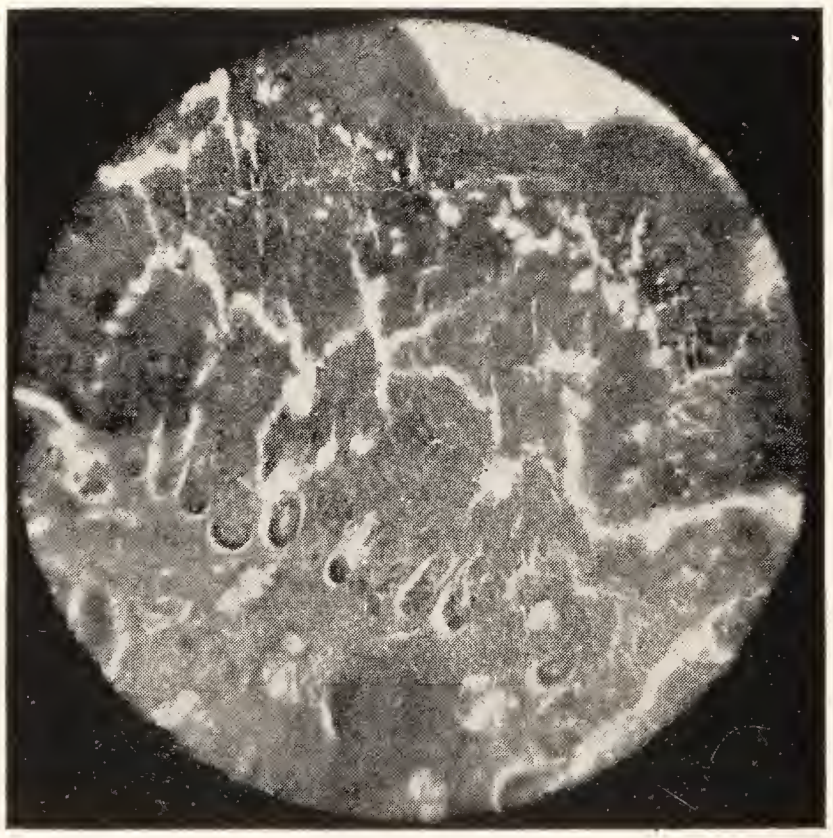
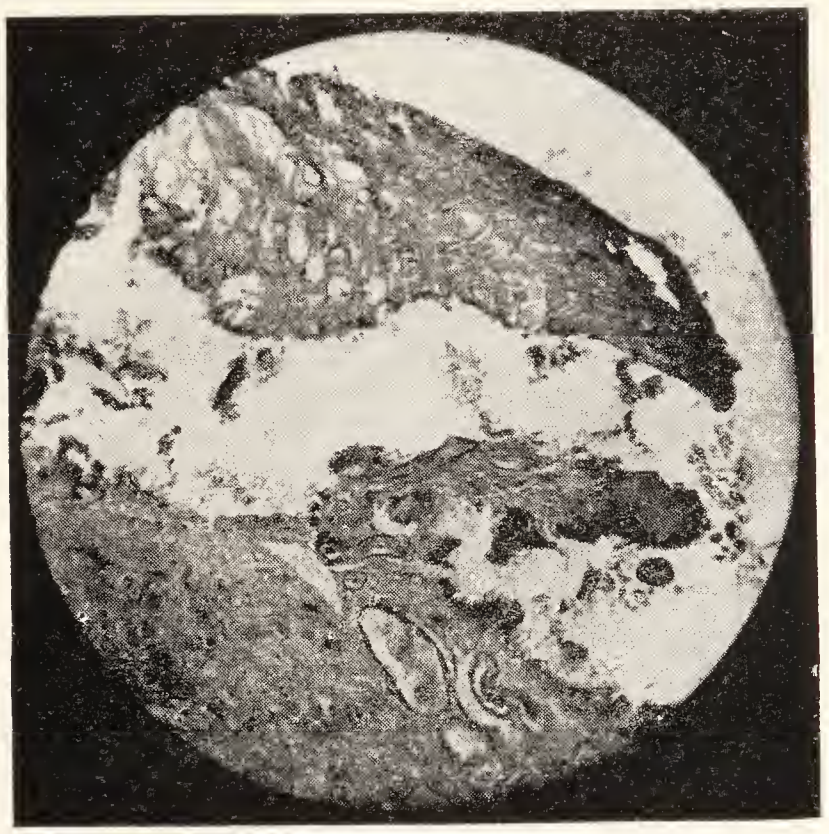


Fig. 13. Diphtheritic dysentery, showing fibrinous infiltration of almost entire thickness of mucous coat.



(*a*)

Fig. 14. Showing sloughing and undermining of the mucous coat.

(*a*)

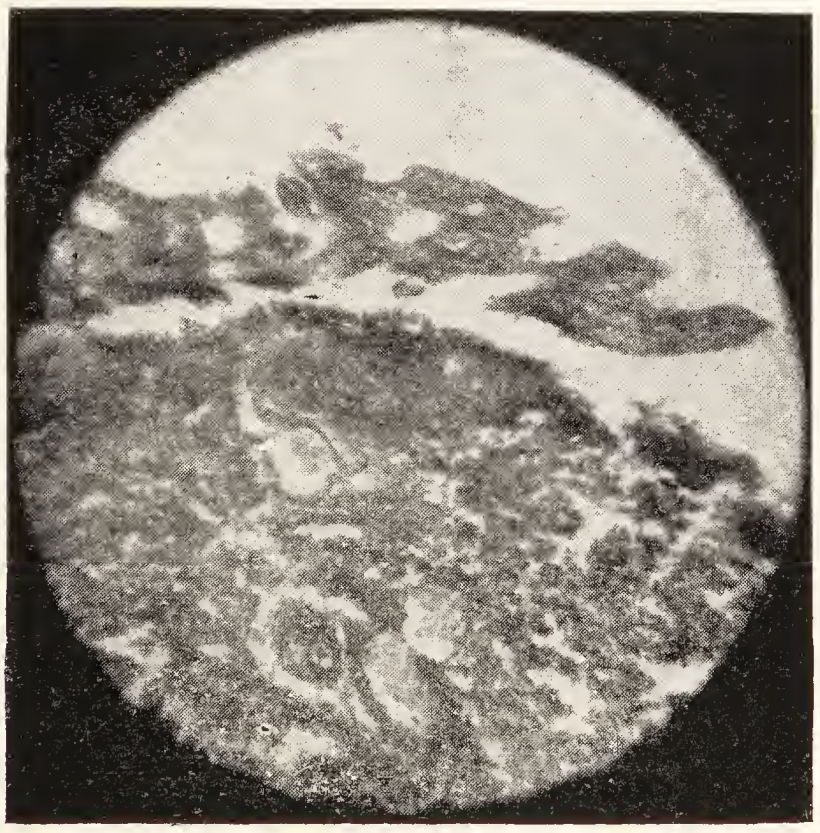


Fig. 15. Showing sloughing and undermining of mucous coat.

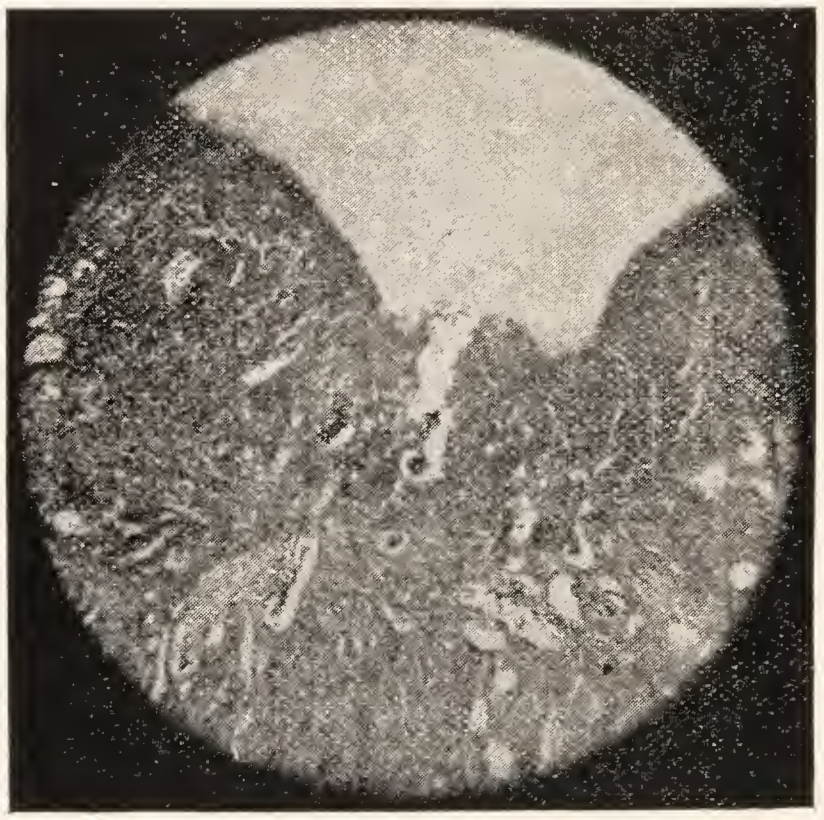


Fig. 16. Showing sloping borders of ulcer.

(*a*)

Fig. 17. Showing fringe of mucous coat (*a*) overhanging edge of ulcer.

prominence owing to extensive ulceration and cicatrization of surrounding parts. Sometimes they are covered by exuberant granulations giving them the appearance of a blackberry.

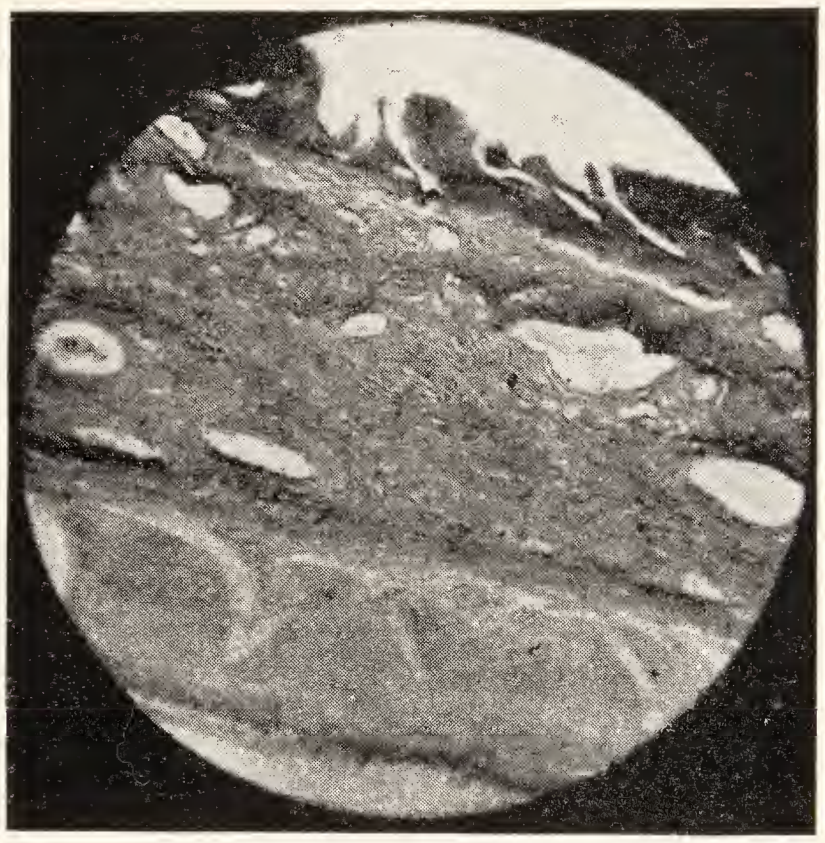


Fig. 18. Showing (*a*) fibrous conversion of submucous coat.

Swollen and sodden granulations from the floors of ulcers may also give this polypoid condition to different parts of the colon.

TABLES

Summary of Post-Mortem Appearances in 80 Cases.

No. of Occasions	Lung	No.	Liver	No.	Kidneys	No.	Glands	No.	Stomach	No.	Small Intestine	No.	Large Intestine
3	Right basal pneumonia.	2	Abscess.	35	Cirrhotic.	18	Mesenteric glands enlarged.	1	Stomach congested.	14	Congested.	2	Old cicatrices.
...	...	...	...	1	Thrombosis of vein.	1	Parotid gland. Metastatic abscesses.	...	...	7	Erosion and slight ulceration.	7	Sloughing condition of mucous membrane.
1	Interstitial pneumonia.	...	...	...	...	...	...	...	...	...	...	5	Croupous and diphtheritic patches on mucous membrane.
5	Catarrhal pneumonia.	...	...	...	...	...	...	...	...	...	...	4	Gangrenous condition.
3	Septic patches of consolidation.	...	...	...	...	...	...	...	...	...	...	1	Follicular.
1	Double acute pleurisy.	...	...	...	...	...	...	...	...	..	...	1	Localised peritonitis.
13	Congestion.	..	...	...	...	...	...	...	...	...	...	1	Perforation.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
1	Emphysema and slightly œdematous.	Normal.	Fatty. Gall stones, and one in the duct.	Fatty.	...
2	Old pleuritic adhesions.	...	Abscess in left lobe containing 1½ pints pus.	Cystic.	...
3	Normal.	Normal.	Pale.	...	...
4	Right pleura adherent and lung hepatized. Left lung œdematous. Both emphysematous.	Fatty.	Healthy.	Cirrhotic.	...
5	...	...	...	...	...
6	Emphysematous. Left lower lobe congested and œdematous.	Puckering and vegetations on mitral. Thickening of tricuspid. Arch of aorta and abdominal aorta very atheromatous, rough and vegetations on bony plates. Thrombosis of axillary artery.	Soft and friable.	Left renal vein thrombosed. Extravasations of blood in left kidney; breaking down of portions of same kidney into pus. Right kidney pale.	..
7	Old adhesions at base of left.	Aortic and mitral valves atheromatous.	...	Fatty.	...
8	Healthy.	Healthy. Considerable atheroma of aorta for age of patient.	Healthy.	Healthy.	Large and soft.

Glands	Stomach	Small Intestine	Large Intestine
...	...	Normal.	Colon extensively ulcerated, especially in vicinity of cæcum.
..	...	...	Numerous ulcerated patches containing sloughs all over the colon.
...	...	Lower portion congested.	Congested with superficial erosions.
...	...	Congestion and some tumidity in lower end of ileum.	Cæcum congested, ulceration slight in descending colon and increasing in extent towards termination of gut.
...	...	Healthy.	Cæcum and colon congested, with extensive ulceration.
...	..	..	Congestion and ulceration from cæcum to rectum.
...	...	...	Acute inflammation of entire colon.
Mesenteric glands enlarged and reddened.	Healthy.	Healthy.	From cæcum to rectum there is an acute congestion with erosion and ulceration. In some places, especially in cæcum and sigmoid, there is a diphtheritic condition round the ulcers. The inflammation is acute, general and not advanced as regards secondary changes.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
9	Fibrinous deposit on both pleuræ. Both lungs congested and œdematous.	Mitral valve atheromatous. Aorta very atheromatous.	Fatty.	Cirrhotic.	Healthy.
10	...	...	...	...	...
11	Both lungs very emphysematous. Calcareous nodule in lower lobe of left and another one in upper part of right lower lobe.	...	...	Cirrhotic and cystic.	...
12	Both emphysematous and bases congested.	Vegetation on aortic valve.	Soft small abscess towards posterior surface.	...	...
13	Both emphysematous and œdematous.	...	Congested.	Congested.	...
14	Left pleura adherent. Left lung congested. Calcareous nodule.	...	...	...	...
15	Commencing pneumonia of right lower lobe.	Roughening of aortic and mitral valves. Aorta atheromatous.	Fatty. Two gall stones.	...	...
16	Lower lobes congested.	Vegetations on mitral valve.	Fatty.	Cirrhotic and cystic.	...
17	Commencing basal pneumonia of right lung.	...	...	...	...

Glands	Stomach	Small Intestine	Large Intestine
...	...	...	Colon intensely congested and covered with numerous small ulcerations.
...	...	Ileum injected towards lower part.	Cæcum and ascending colon thickened and almost denuded of mucous membrane.
...	...	Lower portion of ileum congested.	Cæcum denuded of mucous membrane ; colon congested, red and much ulcerated especially in lower part.
...	...	Healthy.	Walls softened and tear readily. Mucous membrane is thickened and shreddy and separates easily from the other coats, and in cæcum and colon there are several large ulcers.
...	...	Two ulcers in small intestine above ileo-cæcal valve.	Granular and velvety looking in its entire length.
...	...	Congestion of ileum.	Congestion and ulceration of mucous membrane, with here and there diphtheritic patches.
...	...	Two intussusceptions.	Congestion and slight ulceration of ascending colon.
...	...	...	Denuded of mucous membrane to a great extent throughout its entire length.
...	...	...	Thickened and extensively ulcerated.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
18	Old pleuritic adhesions on the right.	Hypertrophied and fatty. Mitral and aortic valves atheromatous.	Fatty.	Cirrhotic and cystic.	Hard.
19	...	Mitral valve thickened.	Fatty.	Capsules slightly adherent.	...
20	..	...	...	Congested.	...
21	Bases of both congested and œdematous.	Right ventricle dilated. Aorta atheromatous.	Fatty.	...	...
22	Lungs emphysematous.	Old pericarditis. Left ventricle hypertrophied. Mitral valve thickened and calcareous. Aorta the same.	Fatty.	Cirrhotic.	Healthy.
23	Lower lobe of right lung œdematous.	Warty vegetations on mitral valve.	Fatty.	Cirrhotic.	...
24	...	Aorta very atheromatous.	...	...	...
25	Base of right lung very œdematous.	Mitral valve thickened. Aorta atheromatous.	...	...	...
26	...	Heart very fatty.	Fatty.	..	...
27	...	...	Friable.	...	...
28	...	...	...	...	...

Glands	Stomach	Small Intestine	Large Intestine
...	...	Congested in several places.	Thickened and extensively ulcerated.
..	.	...	Mucous membrane thickened and congested.
.	...	...	Cæcum deeply congested as also colon, which exhibits numerous ulcers.
...	...	...	Colon congested but no actual ulceration.
Healthy.	Healthy.	Healthy.	Colon deeply congested, eroded and ulcerated in parts. Cæcum purple-red and shows ulcers. Ascending colon eroded and congested. Transverse colon shows small isolated ulcers. Descending colon and sigmoid more advanced ulceration, especially the lower part which is of a deep purple hue. Over the entire mucous surface there is a tenacious slimy mucus mixed with yellow fæcal matter.
...	...	...	Cæcum and ascending colon thickened and ulcerated.
..	...	...	Extremely thickened and extensively ulcerated.
...	...	. .	Rectum thickened and mucous membrane rough and injected. Descending colon irregular ulcers with hard, thickened borders.
..	...	...	Colon soft and tears easily. In a sloughing condition.
.	...	..	Much thickened. Mucous membrane injected and rough and in a sloughing condition in cæcum.
..	...	Last foot of ileum congested.	Entire colon and rectum thickened, intensely congested, irregular on surface and ulcerated.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
29	...	...	..	...	...
30	...	...	...	...	...
31	Old adhesions in both pleuræ.	Heart enlarged and fatty. Left ventricle hypertrophied.	...	Cirrhotic.	...
32	Old adhesions in right pleura. Congestion of base of right lung.	Healthy. Aorta atheromatous.	Healthy.	Healthy.	Large and soft.
33	Slight pleuritic adhesions left apex.	Mitral and aortic valves much thickened.	...	...	Soft.
34	Slight adhesion at right apex.	Healthy.	Large and soft.	Healthy.	Large and soft.
35	Old adhesions in both pleuræ. Both lungs œdematous.	Flabby.	Fatty.	...	...
36	Old pleuritic adhesions. Bronchi thickened. Septic patches of pneumonia in left base.	Muscular substance fairly healthy. Mitral valve thickened and atheromatous. Aortic valve and beginning of aorta atheromatous.	Fatty.	Congested.	Enlarged and softened.
37	...	..	...	...	...
38	..	...	...	Enlarged and slightly cystic.	...

Glands	Stomach	Small Intestine	Large Intestine
..	.	...	Descending colon ulcerated. Rectum thickened, mucous membrane gangrenous.
...	...	Last foot of ileum inflamed.	Colon inflamed and ulcerated.
...	...	Lower portion of ileum congested and ulcerated.	Cæcum and ascending colon much thickened and congested. Large patches of ulceration.
Enlarged and softened.	Diphtheritic patches at opening of œsophagus into stomach.	Lower portion congested and small ulcers.	Large slough on ileo-cæcal valve. Cæcum and ascending colon numerous small ulcers. Descending colon areas of patchy congestion.
...	Dilated.	..	Thickened greatly, large sloughs and ulceration of all degrees.
Mesenteric glands enlarged.	...	...	Thickened, congested and large sloughs of mucous membrane.
Omentum adherent to abdominal wall.	...	...	Red and ulcerated, appearing as a granular surface.
Mesenteric glands soft and red.	Slightly congested.	Healthy. Peyer's patches picked out with pigment.	Entire large bowel seat of ulcerative inflammation. Cæcum deeply congested and eroded. Transverse colon shows large ulcers and muscular coat is exposed. Descending colon and sigmoid are more affected, and the ulceration in places has extended to peritoneal coat and the bowel is so thin it tears on removal. Some evidence of limited peritonitis. The entire mucous coat has a bluish-black appearance, with redness round the ulcers.
...	...	...	Mucous membrane of entire colon ulcerated, softened and of a greenish colour.
...	...	...	Sigmoid and rectum congested. Colon ulcerated.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
39	Normal.	Healthy.	...	Slightly fatty.	...
40	Right lung shows catarrhal patches at base. Left lung is carnified. Apex shows fibroid changes.	Healthy. Atheroma of aorta and at orifice of coronary arteries.	Healthy.	Slightly cirrhotic.	Healthy.
41	Old pleuritic adhesions in both. Bronchitis and emphysema, with some œdema at bases.	Old pericarditis. Thickening of mitral valve.	Healthy.	Healthy.	Healthy.
42	Old adhesions. Lungs slightly emphysematous.	Healthy.	Gall bladder distended with gall stones, one of which obstructs common duct. Perihepatitis. Fatty.	Fatty, and capsules adherent.	...
43	...	...	...	...	...
44	A few emphysematous bullæ.	...	...	..	...
45	Old cicatrices.	...	...	...	...
46	Old pleuritic adhesions. A few inspissated nodules in apex of right lung.	Thickening of mitral and aortic valves.	Small. Capsule thick.	Cirrhotic and cystic.	...
47	Old pleuritic adhesions in both. Both lungs congested and œdematous. Bronchitic.	Coronary arteries, aorta and aortic valves atheromatous.	Fatty.	Cirrhotic and cystic.	..

Glands	Stomach.	Small Intestine	Large Intestine
...	...	...	Cæcum and sigmoid flexure congested and ulcerated.
Enlarged.	Healthy.	Healthy.	From cæcum to rectum shows an acute ulcerative condition. Cæcum and sigmoid are deeply congested, and ulceration is more marked here than elsewhere. The ulcers are more discrete in transverse colon.
...	...	...	Descending colon, sigmoid and rectum congested and ulcerated.
..	...	...	Entire mucous membrane is congested; ulceration of cæcum and first part of rectum.
...	...	...	Mucous membrane deeply congested. Ulceration in first part of rectum and in cæcum.
...	...	...	Congestion and ulceration in first part of rectum and in cæcum.
...	...	...	Entire mucous membrane acutely congested, numerous ulcers.
..	...	Lower end congested.	Mucous membrane congested, thick and rough, most marked in cæcum and lower portion of rectum. Deposit of fibrin on the roughened surface.
...	...	...	Small superficial ulcers in cæcum (which is dilated), and in hepatic flexure of colon, beyond which calibre of gut is much contracted, and in sigmoid and rectum mucous membrane is thick, rough and exhibits many deep ulcers from size of sixpence to split pea.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
48	Lungs — slight emphysema of anterior margins.	Healthy.	Fatty.	Healthy.	...
49	Old adhesions in both pleuræ. Bronchitis. Interstitial pneumonia.	Healthy.	Healthy.	Congested.	...
50	Old adhesions in both pleuræ. Congestion and œdema of both bases.	Healthy.	Fatty.	Fatty.	Firm.
51	Old adhesions of pleuræ, emphysema and chronic bronchitis.	Aorta atheromatous. Wall of right ventricle thin and soft.	Gall stones. Liver fatty.	Cirrhotic.	...
52	Both pleural sacs obliterated. Bronchitis and some collapse.	Healthy.	Soft and friable.	Slightly cirrhotic.	..
53	Old adhesions in both pleuræ.	Dilated and flabby.	...	Cirrhotic	Ulcerative cystitis. Paraplegia.

Glands	Stomach	Small Intestine	Large Intestine
...	...	Lower portion congested and superficially ulcerated.	Mucous membrane of cæcum and adjourning portion of colon congested and superficially ulcerated. In descending colon, sigmoid and rectum there is extensive denudation of mucous membrane, and wall of gut is of a greenish colour.
Mesenteric glands enlarged.	A small ulcer towards cardiac end.	Deep patchy congestion throughout.	Congested throughout, most marked at flexures; and from the splenic flexure to rectum scattered ulcers with ragged, undermined edges.
Mesenteric glands enlarged.	...	...	Ascending and transverse colon distended. Rectum—a large number of round, superficial ulcers, increasing in depth and extent towards sigmoid, just above which ulceration extends to peritoneal coat, which is covered by recent fibrinous exudation. Ulceration gradually diminishing towards hepatic flexure, which exhibits two large ulcers. Cæcum and ascending colon are intensely inflamed.
...	...	Lower portion of ileum congested.	Cæcum congested and superficially eroded. Hepatic flexure is thickened and eroded. This condition of thickening and erosion continues down to sigmoid, where there is thickening but no erosion. In rectum down to a couple of inches above anus the mucous membrane is acutely congested, thickened and deeply ulcerated.
...	Stomach deeply congested where transverse colon is adherent.	...	Peritonitis matting together stomach, transverse and descending colon, left kidney and some coils of small intestine. Distension of bowels. Mucous membrane of rectum is thickened and ulcerated, the ulcers increasing in size till in the sigmoid, mucous membrane is almost absent. At splenic and hepatic flexures peritoneal coat alone remains. The cæcum and ascending colon thickened but otherwise healthy.
...	..	...	Sigmoid and rectum congested and slightly ulcerated. Patches of congestion elsewhere in colon, but otherwise it is healthy.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
*54	Old adhesions in both pleuræ. Congestion of both bases.	Healthy.	Gall bladder one stone. Liver healthy.	Congested.	Congested.
55	...	...	...	Slightly cirrhotic.	...
56	Old adhesions in both pleuræ. Bronchitis and catarrhal pneumonia.	Healthy.	Fatty.	Fatty.	..
57	Bronchitis and emphysema.	Mitral valve thickened. Slight atheroma of arch. Heart fairly healthy.	Nutmeg.	Healthy.	...
58	Left pleuræ obliterated; right adhesions at base and apex. Lungs healthy.	Mitral thickened. Heart healthy.	Nutmeg. Congestion.	Cirrhotic.	...
59	Some old pleuritic adhesions. Congestion and œdema of lungs.	Aorta atheromatous. Thickening of mitral valve.	...	...	...

Glands	Stomach	Small Intestine	Large Intestine
...	...	...	Acute inflammation and œdema of mucous membrane of entire colon going on to superficial necrosis, and the surface is covered by an ash-grey membrane which on removal leaves a raw surface. The rectum and lower half of colon exhibit this superficial necrosis all over.
...	...	...	Mucous membrane from cæcum to rectum thickened and ulcerated. The ulcers, though nowhere deep, are deepest at hepatic and sigmoid flexures.
...	..	Lower portion — mucous membrane congested, and there is a small ulcer with ragged edges 9 in. from valve.	Mucous membrane of rectum is thickened, congested and superficially eroded. Congestion is more marked in sigmoid, and at splenic flexure ulceration is deeper. Elsewhere congestion and ulceration are patchy.
Mesenteric glands enlarged.	...	Small intussusception about $2\frac{1}{2}$ ft. above valve. Taenia solium in small intestine.	Mucous membrane of entire colon and rectum is congested, of a violet colour and superficially eroded. From the splenic end downwards it is thickened. Ulceration is deepest at upper part of rectum, and congestion most marked in cæcum, sigmoid and rectum.
Mesenteric glands enlarged.	...	Last two ft. of ileum congested.	From about six inches below cæcum mucous membrane of colon and rectum is congested, violet in colour and thickened with some half dozen deep ulcers with ragged, undermined edges. Thickening and congestion more marked in rectum and sigmoid where there is extensive superficial erosion.
Mesenteric glands enlarged.	...	...	Mucous membrane of large gut inflamed, thickened, eroded and granular looking. Rectum, lower portion of sigmoid and cæcum deeply congested, and in the latter are small ecchymoses.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
60	Both pleural cavities obliterated. Small calcareous nodule in apex of left lung. Both lungs rather small and atrophied.	Aortic arch atheromatous. Mitral valve thickened. Muscular substance soft and thin, and at apex has almost disappeared.	Fatty.	Cirrhotic.	...
	Right pleural cavity obliterated. Both lungs congested.	Mitral and aortic valves atheromatous. Heart very fatty.	Fatty.	Healthy.	Large.
*62	Left pleural cavity obliterated. Congestion and œdema of both lungs, and catarrhal pneumonia of right.	Old adhesive pericarditis. Heart greatly enlarged, fatty. Thickening of aortic and mitral cusps. Arch of aorta atheromatous.	17 gallstones. Liver enlarged and fatty.	Cirrhotic and cystic.	Large, soft and pulpy.
*63	Slight pleural adhesions at right apex. Bronchitis.	1 oz. fluid in pericardium. Mitral and aortic cusps thick. Arch of aorta atheromatous. Wall of right ventricle is very thin.	Fatty liver.	Cirrhotic and cystic.	Firm and very dark.
64	...	...	Fatty	Congested.	Soft and pulpy.

Glands	Stomach	Small Intestine	Large Intestine
...	...	...	Mucous membrane of rectum is pale and eroded. Erosion increased in sigmoid and from there to the cæcum there is gradually increasing congestion, which assumes a violet hue.
Mesenteric glands enlarged.	...	...	Sigmoid greatly distended. From cæcum to splenic flexure mucous membrane is thickened and shows numerous ulcers from a pea to a shilling in size, and exhibits yellow sloughs, with sharply-depressed edges. Next two feet fairly healthy but show areas of congestion. Lower part of sigmoid is thickened and has numerous small ulcers.
Mesenteric enlarged.	...	Lower portion of ileum congested and eroded.	Mucous membrane of colon has been extensively destroyed by ulceration, and what remains is black and deeply pigmented. Large, irregular and deep ulcers in cæcum, and sigmoid and mucous membrane at anus is thickened and deeply ulcerated.
Mesenteric glands enlarged.	...	Large scrotal hernia, which has a gangrenous look.	Upper part of colon is thickened, œdematous and extensively ulcerated. In descending colon and sigmoid ulceration but less thickening. Rectum is pale, thickened and eroded. Throughout colon there are patches of intense violet congestion.
..	...	Congested and superficial erosion of lower portion of ileum.	Large bowel shows deep and extensive ulceration, and in the transverse colon there is almost perforation.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
65	Healthy.	Left ventricle hypertrophied. Mitral cusps and arch of aorta atheromatous.	...	Slightly cirrhotic.	...
66	Pleuritic adhesions at left apex, which has a few caseous nodules. Bases of both lungs œdematous.	Heart hypertrophied. Aortic and mitral cusps atheromatous.	Enlarged and cirrhotic.	Cirrhotic.	...
67	Adhesions at both apices	Heart healthy. Slight atheroma of arch of aorta.	...	Slightly cystic and cirrhotic.	Enlarged and flabby.
*68	Pleuritic adhesions to diaphragm (right). Bronchitis and emphysema.	Left ventricle hypertrophied. Mitral cusps atheromatous and contracted.	...	Slightly cirrhotic.	...
69	Slight adhesion right apex. Bronchitis. Congestion of right lung.	Heart enlarged. Slight atheroma of arch of aorta.	One large gall stone.	Cirrhotic and hydronephrosis.	Healthy
70	Old adhesions in left.	3 oz. fluid in pericardium. Heart small.	...	Cirrhotic.	..
*71	Slight adhesions at both apices. Middle lobe of right lung shows small yellow patches of septic pneumonia, which are soft and breaking down.	Atheroma of arch of aorta.	Large and fatty.	Cystic and cirrhotic.	..

Glands	Stomach	Small Intestine	Large Intestine
...	...	...	Mucous membrane of colon is deeply congested throughout, and lower part of sigmoid is thickened and extensively ulcerated, of a dark grey colour and gangrenous. Cæcum is congested and ileo-cæcal valve is ulcerated.
...	...	...	Mucous membrane extensively ulcerated and thickened, most marked in descending colon, where it is grey, black and gangrenous. Cæcum is congested and ulcerated.
...	...	...	Mucous membrane of descending colon of a bluish colour, thickened and extensively ulcerated. At lower end of sigmoid a constriction from contraction of partially healed ulcers. Cæcum congested and exhibited old cicatrices. The rest of the colon shows patchy areas of congestion and numerous ulcers.
Enlarged mesenteric glands.	...	..	Cæcum shows slight congestion which increases further down till in descending colon mucous membrane is red, rough and granular looking. In lower part of sigmoid there are large solitary ulcers, edges undermined with infiltration of surrounding mucous membrane.
...	...	Two patchy areas of congestion in lower portion of ileum.	Cæcum acutely congested. Colon shows areas of patchy congestion, and in splenic flexure and sigmoid it is of a greyish green colour and is eroded and granular.
...	...	...	Congestion of cæcum and sigmoid, where also there is slight superficial ulceration.
..	...	Last six inches of ileum deeply congested.	From ileo-cæcal valve down to anus interior of intestine is of a greenish grey colour and deeply and extensively ulcerated. Sigmoid is adherent to abdominal wall and tears on removal, the muscular coats have disappeared and peritoneal coat is thickened in the descending colon.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
†72	Some old and recent adhesions in right pleura. Small patches of septic consolidation, size of a sixpence, in lower lobes, which are yellow and breaking down. The rest of the lungs are œdematous.	Arch of aorta atheromatous. One small vegetation on aortic cusp.	Healthy.	Healthy.	...
73	Left lung collapses. Right fairly healthy.	Pericardium distended. Right ventricle thin and dilated, left ventricle hypertrophied. Mitral stenosis Dilatation and atheroma of arch of aorta.	Healthy.	Cirrhotic.	..
74	...	...	Soft.	Congested.	...
†75	Old adhesions of right pleura. Lungs healthy.	Small, soft and flabby.	Healthy.	Healthy.	...
†76	Obliteration and great thickening of left pleura. Adhesions in right. Left lung disseminated nodules of tubercle and cavities in apex. Right lung fairly healthy; a few nodules in apex.	Fatty.	Fatty.	Firm but not waxy.	...

Glands	Stomach	Small Intestine	Large Intestine
Pyæmic abscesses in parotid gland.	...	...	Patchy congestion from cæcum downwards. In sigmoid there are small ulcerations and a patch of the mucous membrane is in a gangrenous condition, as also in cæcum and ascending colon.
...	..	...	Cæcum congested and shows a few ulcers. Deep congestion of ascending, transverse and descending colon, with prominence of the follicles which are beginning to ulcerate. A few small ulcers in rectum.
...	...	...	Congestion of cæcum and colon, the latter exhibiting numerous ulcers ; a few ulcers only in cæcum.
Mesenteric glands enlarged and soft.	...	A few of the lowest Peyer's patches picked out with black pigment.	Entire mucous membrane of colon and rectum is in a state of sloughing ulceration, commencing abruptly at ileo-cæcal valve. Cæcum and ascending colon show yellowish white sloughs of mucous membrane, and one in transverse colon is $1\frac{1}{2}$ inches in diameter. Mucous membrane between the sloughs and ulcers is in a state of intense congestion and covered with blood-stained mucus. Abundant sub-mucous hæmorrhages.
...	...	A few tubercular ulcers in small intestine.	Is greatly thickened and contracted, the thickening being chiefly confined to mucous and sub-mucous coats. Mucous membrane is rough and granular, with extensive superficial ulcerations.

No. of Case	Lungs	Heart and Vessels	Liver	Kidneys	Spleen
†77	Encysted effusion in lower part of right pleura. Old adhesions in both. Chronic thickening of bronchial lining.	Pericardial cavity obliterated. Heart flabby. Right ventricle dilated. Slight atheroma of aorta.	Healthy. Gall bladder contains 1 gall stone.	Healthy.	...
†78	Healthy.	Small mitral valve atheromatous and puckered d. Aorta atheromatous.	Congested.	Cystic and markedly cirrhotic.	Healthy.
79	Patchy, fibrinous exudation on pleuræ. Catarrhal pneumonia in both lower lobes, more extensive in right.	Enlarged. Mitral valve incompetent. Atheroma of arch.	Fatty.	Cirrhotic.	Soft.
80	Right lung has some catarrhal pneumonia in lower lobe.	Heart slightly hypertrophied. Aortic segments have warty vegetations and are incompetent. Aorta atheromatous.	Cyanotic atrophy, fatty, and is the seat of some small syphilitic gummata.	Right kidney is large and cirrhotic. Left one is very small and markedly cirrhotic.	Healthy.

* indicates a

† indicates a

Glands	Stomach	Small Intestine	Large Intestine
Mesenteric enlarged.	...	Mucous membrane, lower portion of ileum of a deep purple colour, and last few inches superficially eroded.	Lymphy exudation on peritoneum in neighbourhood of cæcum. Mucous membrane of cæcum is of a brownish colour and eroded. Ascending and transverse colon show discrete, well marked ulcers, and mucous membrane generally is of a rusty brown hue, with acute congestion of the folds. Sigmoid and rectum of a dark brown colour and mucous membrane extremely eroded and ulcerated. Rectum and lower portion of sigmoid thickened and œdematous. Transverse and descending colon show isolated sloughs of mucous membrane.
Healthy.	Healthy.	Lower few inches of ileum congested.	Entire large bowel acutely inflamed, with an extensive diphtheritic exudation which is firmly adherent and brings away the mucosa on removal, having a raw red surface. Areas of erosion where the exudation has separated in ascending and descending colon. Cæcum is intensely congested, almost bluish-black in colour.
Healthy.	Healthy.	Healthy.	Slight ulceration on the colic side of the segments of the valve. Patches of congestion in cæcum and colon. In sigmoid there are cicatrices from formerly healed ulcers. The edges are thickened and in some inflamed. The rectum is puckered and thickened from former ulceration, and is of a dirty yellow colour. The interior of bowel is coated with mucus, and fæcal contents are bright green in colour.
Enlarged and softened mesenteric glands.	Healthy.	Healthy.	Large intestine shows a general ulcerative colitis, most marked in lower sigmoid and rectum, which are thickened, have lost their normal appearance and are of a dirty greenish colour and covered with sticky mucus. The coats are œdematous and tough.

case described.

coloured plate.

CHAPTER V

ÆTIOLOGY

In the chapter dealing with the association of dysentery with other diseases, its ætiology has been incidentally touched upon, and this one will, to a certain extent, contain recapitulatory matter. The number and variety of causes said to give rise to dysentery indicate that some of them at least are to a large extent accidental, and capable only of aggravating, not of causing it. To others, however, more value must be attached; and, until bacteriology proves conclusively that the disease in question is due to a definite organism capable of artificial cultivation, and on inoculation of reproducing dysentery in its entirety, we are entitled to consider them, at least to all intents and purposes, as causes. We must remember, however, that the mere presence of the organism may not of itself be sufficient to occasion the disease.

As in phthisis, so in dysentery, many are exposed to the contagion and yet escape. We have seen that dysentery has been closely associated in the past with typhus fever, malaria, purpura and scurvy; and in proportion as the cause or causes of these affections have been recognised and banished, it has, along with them, disappeared or diminished.

The introduction of sanitation and improved agriculture have been the means of abolishing the two former, and the recognition of the important part played by dietetics in the maintenance of a healthy condition of the economy, has made scurvy, at least, a thing of the past.

On reviewing the circumstances under which dysentery

arises, we find that—when associated with typhus fever—overcrowding and insanitary conditions generally, impure drinking water and enfeebled general nutrition from impoverished and unwholesome diet, have always been present.

The same may, in great measure, be said of the conditions under which it co-exists with scurvy. The over-crowded, ill-ventilated forecastle, the damp atmosphere and unhealthy exhalations from the occupants, prove very favourable—especially when combined with contaminated drinking water and bad food—for the production of dysentery alone or in combination.

In typhus, dysentery and scurvy, then, certain conditions are invariably present, and necessary for their production. We find that the first two diseases, when epidemic or endemic, are almost invariably associated with overcrowding, but that scurvy is not necessarily so, outbreaks of it occurring where no blame could be attached to insanitary surroundings. By a further process of analysis, and having regard to what has already been said as to the conditions present when dysentery and scurvy exist, we find that both these diseases are dependent upon unwholesome and defective food supply, special importance, however, attaching in the case of dysentery to overcrowding and contaminated drinking water, neither of which causes are essential to an outbreak of scurvy. Beyond mentioning bad food as a causative element in the production of dysentery, no detail has been entered into, but in this connection there are one or two points worthy of notice to which brief allusion may be made. In many asylums, pork, which at different times forms a considerable item in the patients' dietary, has been credited with being the cause of the dysentery, and discontinued, but without obvious result. The supposition was a random one, hazarded, so far as I can make out without any idea of a possible connection existing between it and swine fever, and certainly before the similarity in the pathological lesions of the two affections was recognised. We find, moreover, that the same

unsupported statement has been made with reference to an outbreak occurring among soldiers abroad. On the whole, I think the bulk of the evidence indicates that the mere consumption of pork bears no, or only an accidental, relationship, such as might apply to any other article of diet. If the pork was supplied from an animal dead of swine fever, or was tainted from commencing decomposition, the symptoms and lesions, I think, would be more those of an irritant poison than of ulcerative colitis, which, as regards lesions, is limited to the colon. In this connection it will be interesting to see the results of the proposed experiments of feeding and inoculating pigs with the products of ulcerative colitis from human beings.

Another point to which I wish to refer regarding the influence of diet in the causation of dysentery in asylums is the supply of bread and potatoes. These staple articles of food, more especially the latter, have, when diseased, often been shown to predispose to dysentery, and as, in addition, a scorbutic taint may be induced, it is necessary that great attention should be paid to selecting those of the best quality.

Another important factor again in the causation of dysentery is malaria. Now, we know that this disease depends upon the presence of marshy land where the water is stagnant and saturated with products of organic decomposition. It is a local (endemic) cause, and disappears on cultivation and drainage of the soil.

Allusion has already been made to the important part played by organically impure water in the causation of dysentery in temperate climates, and numerous instances might be cited where it directly owed its origin to this cause. Now, the drinking water in malarial districts, and indeed in the Tropics generally, is notably bad, and would in this country be unhesitatingly condemned as unfit for use. With these facts prominently before us, would it not be legitimate to consider the miasmatic poison, alleged to be causative of dysentery, as really consisting of a

contaminated water supply? So impressed have I been with this, that I strongly incline to the belief that endemic tropical dysentery of malarial origin will eventually, to a great extent at least, be found dependent upon water contamination.

Viewing now the ætiology of dysentery from another aspect, a bacteriological one, we infer from the names pseudo-membranous, croupous and diphtheritic, as applied to the anatomical appearances which it presents, the manifest tendency to regard it as a specific disease due to some form of micro-organism. I have personally been unable to identify the disease with the invariable presence of a definite organism owing to my want of practical acquaintance with the methods of bacteriological research. Dr Goodliffe, the pathologist, has, however, kindly supplied me with the results of his investigations in this connection, and to them I shall refer later on; meantime, what I have to say is taken from the labours of others.

We know that at birth the intestinal contents are sterile (though cases have been recorded of intra-uterine infection with enteric fever), but speedily become infected with hosts of micro-organisms, chiefly by ingestion of food. In this connection it is interesting to note that milk supplied from town byres is stated to contain over 300,000 per cent. of micro-organisms. The majority of the organisms introduced along with the food are speedily destroyed by the gastric juice, so that, comparatively speaking, a small proportion survive in the intestines, where they play, according to some, an important part in digestion. The number varies with the nature of the food, but many are so uniformly to be found in the fæces and adherent to the internal surface of the bowel, that they may be considered as normal inhabitants. To some of these, pathogenic properties are ascribed, and well-known pathogenic organisms are frequently found as temporary and even harmless occupants.

A division into two groups might therefore be adopted:—

I. Organisms normally present.

(*a*) Those which are non-pathogenic.
(*b*) Those said to possess pathogenic properties.

II. Organisms not normally present in the contents of the bowel, some possessing indisputable pathogenic properties, and whose presence may or may not occasion the disease attributed to them, and others requiring further confirmation in this respect.

So far as at present concerned, we may dismiss without remark the non-pathogenic organisms and turn our attention to what is known experimentally and otherwise about the other members of the groups.

In group I., sub-division (*b*), the Amœba Coli and B. Coli are the organisms to which, rightly or wrongly, great importance is attached as being the specific cause of dysentery. Let us arrange and examine, therefore, the facts obtained by different observers in their investigations relative to the presence of these organisms as causative of the disease, and see how far they accord with each other.

First, with reference to the Bacillus Coli. We are told that a few days after birth the sterile intestine of the newborn infant contains it in millions, a fact pointing to its universal diffusion through the air. It is, moreover, stated to play an important part in the causation of dysentery, and also to set up ulcerative colitis (by some considered quite a different affection from dysentery) in the human subject. Inflamed piles and osteomyelitis are also said to be occasioned by its presence. This latter affection has been produced experimentally in rabbits, in whom also the B. Coli induced diarrhœa and collapse, and two experimenters, Salvati and Gætano, have prepared a toxin from the culture of the B. Coli, which confers immunity from its lesions. Arnault, in 1894, read a paper before the Société D'Histologie de Paris, in which he says that he had succeeded in isolating the pathogenic organism of Dysenteria Nostras, a bacillus in many respects like the B. Coli Com.

I have so far as I am able, and as briefly as possible,

stated what is known regarding this organism and its relation to diseases which it is said to occasion, and although personally not able to supplement in any way what has already been discovered, still I perceive obvious objections to admitting it as at least the sole cause of dysentery.

First, it appears far-fetched and imaginative to recognise as the cause of a specific disease an organism which exists harmlessly and in myriads in the intestine a few days after birth. Second, it is well known that infants and children are more susceptible to intestinal disturbances than adults, and yet we are asked to believe that the B. Coli produces no evil results in the former, and is the cause of a fatal disease in the latter. Third, it is stated that the B. Coli is capable of setting up inflammation in Peyer's patches, whereas in dysentery these collections of glands are, so far as my experience goes, never affected.

Turning now to the other alleged pathogenic organism, the Amœba Coli, we encounter here, in the contradictory and varying results obtained by different observers, the same want of unanimity which prevented anything like a definite acceptance of the part played by the B. Coli in the causation of dysentery. Some observers consider the cases in which the Amœba is found as different from the ordinary form of dysentery, and use the term Amœbic dysentery as indicative of this type. Tropical dysentery is stated to depend entirely on its presence, although other writers consider it as only partly responsible, and call the variety in which it occurs Amœba enteritis. Gasser makes the assertion that the A. Coli is present in considerable numbers in 20 per cent. of healthy stools, and experimentally he has induced dysenteric ulceration by injecting the stools containing the Amœba into the large intestine of a cat. He questions, however, the value to be attached to their presence, as the same result was produced by injecting sterile vegetable *débris*. Quincke and Roos, in their investigations, produced dysentery in cats by injecting into the bowel the stools from Amœbic dysentery. Feeding by

the mouth, though fatal, only caused ulceration when the Amœbæ were encysted. They consider that, both in dysenteric and healthy stools, harmless Amœbæ are also at times to be found. A condition resembling acute nephritis has also been attributed to the presence of Amœbæ and the existence of the A. Coli, and the part it plays in hepatic abscess need only be mentioned.

Briefly reviewing what has been said concerning these two organisms, we find that the B. Coli exists in myriads in the intestinal canal of infants without evil effects, that it is stated to cause dysentery, suppuration of piles, and osteomyelitis in the adult human being, and that the same affections have been produced experimentally in animals.

We are asked, in fact, to credit one organism with the power of inducing several specific diseases differing widely from each other. So far as I am aware, the organisms of other specific diseases, diphtheria, tubercle, erysipelas, are not credited with this property, and why, then, dysentery?

In view, therefore, of the conflicting evidence of experimenters, we must, I think, consider the B. Coli, so far as dysentery is concerned, as playing a secondary and unimportant part. It is found in the healthy bowel in large numbers, and would it not therefore be more rational to attribute its survival throughout prolonged cultivation as due to the fact of its greater hardihood and ability to grow in media, which are not suitable for the life of the other organisms present along with it in the dejecta of dysentery, and among which with different culture media the microbe of the disease may eventually be found?

Summing up with reference to the A. Coli, we are told that it exists in 20 per cent. in healthy stools, and that, in addition to causing a variety of dysentery, hepatic abscess and nephritis are said to be occasioned by its presence. Here again the same objections may be urged as in the case of the B. Coli. The experiments are directly contradictory, and any deduction to be drawn from injecting a pure culture into a

cat's rectum and tying the anus is in my opinion of no value. The resulting swelling and erosions of the mucous membrane would, I think, be as readily produced by the tying of the anus alone.

Coming now to the second group of organisms, we are confronted by two micro-organisms which have claims to be considered as a possible, if not the probable cause of dysentery, one a diplococcus and the other a bacillus. The former does not liquefy gelatine, and is not coloured by Gram's method, and a culture mixed with sterilized water and injected into the rectum of cats and dogs occasions dysentery. Two different bacilli seem to have been specially described. Both are short and slender, but one has scanty power of movement, stains badly, and does not liquefy gelatine. In its physical appearances and mode of growth in gelatine, etc., it is identical with the B. Coli, the only difference appearing in its growth on potato, where it develops as a dry yellow membrane, whereas the colonies of the B. Coli are yellow and juicy. The other bacillus stains with Gram's method, liquefies gelatine, and is very mobile. When cultivated pure, it causes in animals muco-sanguinolent purging, ulceration of the large intestine, and swelling of the mesenteric glands when injected subcutaneously per rectum or swallowed with food. To it is attributed a variety of dysentery occurring in Japan. Here let me state the tendency there seems to be to classify dysentery into varieties or types according to the micro-organisms which have been successfully isolated and cultivated pure. To the diplococcus is assigned a variety, to the Amœba Coli a variety, to a bacillus a variety, and M. de Silvestri believes there exist several varieties of dysentery due to different specific micro-parasites. If these are varieties, then what is the disease? In enteric fever, tubercular and other diseases, the causes are constant. In dysentery, on the other hand, proof as to the existence of a constant cause is yet wanting. Various organisms have apparently caused similar morbid processes productive of a series of phenomena to which we give the

name of dysentery. Why, then, on this account seek to confuse by describing varieties simply because different organisms are capable of producing the same disease? May this variety in the micro-organisms not depend on some climatic or other conditions, favouring the growth of this or that one? Knowing as we do the difficulties attending bacteriological investigations, and the numerous sources of fallacy as evidenced by the directly opposed results of experimenters, this classification then into varieties is not to be commended. Wherein, it may be pertinently asked, do the varieties of dysentery, said to depend on the B. Coli, the A. Coli, a diplococcus and a bacillus, differ from each other, except in the fact that the ultimate issue of cultivation experiments results, in the hands of different observers, in the survival of different micro-organisms. A plunge of a sterilized platinum wire into an ocean of microbes, and thence into a tube of gelatine, and the survival of the hardiest—two of them present in the normal contents of the bowel—determines the specific cause not apparently of dysentery, but a variety. To speak with some exaggeration, new micro-organisms and varieties of those already known are being daily discovered and described. Our daily food is proved to teem with thousands, and the filters, sterilizers, etc., to be used to render it fit for consumption, apart from being entirely beyond the means of the poor, would make even for the wealthy a meal a very laborious undertaking, and probably not so palatable. Severe penalties attached to keeping dirty byres, or stagnant and polluted water in their neighbourhood, selling of diseased meat, etc., will do more to diminish typhoid and other diseases than laying down rules how food is to be rendered safe for consumption, which either could or would not be carried out by one in a thousand. I do not mean to disparage the efforts and discoveries of those who are devoting themselves to the bacterial origin of disease, but merely wish to protest against conclusions being too readily drawn, and against safeguards which are only obtainable by a limited few.

And now let us turn from this perhaps unwarrantable digression and apply the germ theory of disease to dysentery. If it is due to a specific micro-organism, we have to determine whether it already exists in the normal tissues and contents of the bowel, or whether it is introduced from without. If the former, we have to ascertain what conditions favour its development, and these have in measure already been indicated. The view that an organism to whose presence the origin of so many diseases has been ascribed can be the specific cause of the one in question is one which for the reasons already adduced I do not favour. If the latter, is its presence alone, and independent of other co-existing circumstances, all that is necessary to produce the disease? We are aware, to take familiar examples, that the tubercle and typhoid bacillus are both capable of existing in the human organism without causing the respective diseases attributed to them. How often and in what quantities must the former be inhaled and yet not, unless certain other conditions are present, produce tubercular disease, and how often are both water and milk containing the latter organism drunk, and yet the individual escapes typhoid? So far as tubercle is concerned, we are justified in saying that, unless there is a condition of malnutrition, the bacillus, if not at once destroyed, remains innocuous. In typhoid, that Eberth's bacillus attacks those who, so far as our knowledge and observation of them go, are in a state of perfect physical health, and this together with the fact that it may enter with the ingesta and yet not produce typhoid, would seem to indicate that some conditions other than its mere presence are necessary for the production of its specific effect. The same remarks are applicable to the case of dysentery, which, as I have elsewhere fully indicated, is specially liable to attack the feeble and debilitated in overcrowded workhouses and asylums. That it is dependent on some organism I have no doubt, but as yet no conclusion can be arrived at as to the particular one upon which it depends; or else we must admit, from the varying results of different ex-

perimenters, that a definite specific disease is capable of being produced by several different organisms. This apparent anomaly, however, may in time be shown to be due not to different organisms, but to different forms assumed by one and the same organism under different conditions of life and development.

Turning now to the bacteriological investigations of the disease as conducted by Dr Goodliffe for a period of nearly two years, we find that in every instance he has succeeded in isolating a micro-organism which he classifies as a bacillus, and which will be fully described later on. So far, the literature dealing with this aspect of the question is almost *nil*. This, and the fact that he has had no opportunity of practically studying the bacteriology of tropical dysentery, will to a certain extent disparage the evidence when it is sought to prove that this ulcerative colitis is in reality dysentery, or, save the term, a variety of dysentery. I have referred to the organisms previously discussed as giving rise to varieties of dysentery, and the same, I think, may be claimed for the bacillus which Dr Goodliffe has discovered and isolated. He agrees with Silvestri, Ogata and others, who aver that there are several varieties of dysentery, each caused by a different organism.

Concerning his own investigations, Dr Goodliffe says, "The results have certainly led me to believe that the form of ulcerative colitis met with here is due to a specific bacillus, which—1st, is always present in undoubted cases of the disease; and 2nd, is capable of cultivation outside the body on artificial media, thus conforming with two out of the three fundamental conditions necessary to establish its claim as the origo mali." The reproduction of the same disease by inoculating the cultivated bacillus he has unfortunately not been able to verify. The bacillus so closely resembles the B. Coli, that in his earlier investigations he was inclined to believe that it would prove to be one of the many varieties of this organism, being thus in accordance with the view

of Arnaud and others, who hold that dysentery is due to a pathogenic variety of the B. Coli, of which many varieties have been described, all having morphological resemblances, but differing in some reaction or mode of growth. The organism associated with the bowel affection here is, however, quite distinct from any described variety of the B. Coli, moreover, though cultivated through numerous generations on various media (sometimes to the 30th generation), it has always maintained its individuality, and has not reverted to any known type. It has distinct characteristics, reactions and modes of growth, and its claim to be recognised as a distinct organism receives some support from analogy. In the bacillus of enteric fever, for example, we have a well-known organism which has many properties in common with the B. Coli; indeed Roux, Rodet and others go so far as to say that the B. typhosus is only a variety of the B. Coli which has taken on a pathogenic character. The B. typhosus and that of ulcerative colitis appear to stand in a peculiar relationship to each other. Linked, as it were, through the B. Coli, this relationship is carried out in the diseases to which they give rise; for cases of ulcerative colitis are occasionally observed which anatomically, and in some respects clinically, very much resemble typhoid fever, and, as it is known that occasionally the lesions in enteric fever may be limited to the large bowel, to differentiate between colotyphoid and typhoid ulcerative colitis without extraneous aids becomes almost impossible. Dr Goodliffe, however, believes his bacillus to possess such distinctive characters as to enable a diagnosis to be made by bacteriological examination.

The following are the characters according to him of the bacillus of this ulcerative affection of the colon, as found in the evacuations, when isolated and cultivated in artificial media.

It is a short, rod-shaped organism with rounded ends, usually from 1 to 2 μ long and about ·5 μ broad. Often,

however, it is almost as broad as long, and is frequently seen in pairs, so that it looks like a diplococcus. It stains easily with Loeffler's aniline blue and the Ehrlich-Weigert or the Ziehl-Nielson methods. It is not decolourized, or only with great difficulty, by Gram's iodine solution, and this forms one of its chief distinctions from the B. Coli and B. typhosus, both of which part very readily with their colouring matter when treated in this way. It does not liquefy gelatine, is an aërobic and facultative anaërobic bacillus; its anaërobic properties appear to be less pronounced than those of the B. Coli, the deeper colonies in a shake culture developing to a much less extent, and a stab culture also bears this out. In a hanging drop, it appears in certain cases to exhibit independent movements, but Dr Goodliffe expresses doubt as to whether or not this is a constant feature. He has not been able to demonstrate the presence of flagellæ, though frequent and careful examinations have been made by the special methods recommended for their detection. The bacillus does not, he thinks, form spores; none have been observed in the bacillus when cultivated in various media. This conclusion is confirmed by other facts. Artificial cultures, if left in the incubator at a temperature of 48° C. for twenty-four hours, fail to give any development on inoculation of another tube. Spores were also looked for in the following manner. A tube was inoculated, shaken up and put in the incubator at a body temperature for twenty-four hours; the tube was then allowed to stand at ordinary room temperature for a day, put again into the incubator for another twenty-four hours, allowed to stand another day, and finally put in the incubator at a temperature of 48° C. to 50° C. for twenty-four hours. No growth was got on inoculating a fresh tube. This method was resorted to in the hope of encouraging spore formation, as we know that sometimes organisms will multiply by division at a certain temperature and by spores at another. The fact that the organism is so frequently met with in pairs favours the view

that its usual method of generation is by division and not by spores. The bacillus grows most rapidly at a body temperature, colonies developing and clearly showing in twenty-four hours on agar. At any ordinary room temperature they appear in two days. Colonies fail to develop if kept at a temperature much above 45° C. The following are the appearances of growths in the different media. In bouillon it develops well, and causes cloudiness in twenty-four hours at body temperature. In from four to seven days a whitish deposit is seen at the bottom of the tube. In nutrient gelatine in shake cultures, at the ordinary temperature of the laboratory (about 60° F.), colonies develop as minute whitish specks in from thirty-six to forty-eight hours. In three or four days they are well developed, and appear as small glistening white spots like specks of white paint (tube I.). If the colonies are thickly set, there is no further change except an occasional coalescence; if, however, they have room for further growth, they develop into round flat discs with a smooth edge, distinct thickened rim, sharp even border, and often a white spot in the centre (tube II.). A hand lens often shows concentric markings on the rim and a fine granularity of the thinner intermediate parts. After a week or ten days they cease to grow, but maintain their glistening white appearance throughout, even if kept for weeks, thus differing from the colonies of B. Coli, which become brownish. A stab culture in the same medium grows all along the track, and has a yellowish white appearance as seen through the medium, with here and there arborescent branchings. These, however, when examined with a lens are seen to be coarser, more sharply marked, and not so intricate as those of a similarly prepared growth of B. Coli, and the growth along the track is not so abundant. At the surface a spherical raised growth is observed, glistening white in appearance, and not spreading far from the point of inoculation, whereas the colon bacillus spreads considerably over the surface. Occasionally small gas bubbles are seen along the track, but this is not constant.

A surface culture is seen when fully developed in four to six days, is white, sharply demarcated with slightly indented margin where there is a rim of thicker growth (tube III.). On agar much the same appearances are presented. On potato a very distinct creamy yellow growth is obtained, looking moist and slimy, with a sharply marked border. In a slightly acid medium, the growth is profuse, the colonies in a shake preparation being distinct and well marked in twenty-four hours at 60° F. It is only when the medium is distinctly acid that growth fails to take place. In sterilized milk at body temperature there is coagulation of casein in from two to three days. In milk tinged with litmus the bluish colour is turned to pink, and this pink colour is persistent, thus differing from the reaction given with many of Booker's varieties of B. Coli. Indol reaction:—A broth culture is kept at body temperature for three days, and nitrous acid then added with the production of a distinct red colour. The organism will also grow on a nutrient medium which contains ·05 per cent. carbolic acid. If the colonies are exposed to direct sunlight for some hours, they fail to give growths on inoculation. If dried on slides, they also lose their vitality. The bacillus is usually associated in the dejecta with a great many other organisms, mention of which shall be made later. In some cases, however, the discharges exhibit almost a pure growth, a possible explanation being that the other organisms are suppressed by the great vitality of the specific one. Shenk enunciated this view with regard to cholera; and Dr Goodliffe informs me he occasionally got in cases of enteric fever almost pure cultures. The organism is also present on the mucous lining of the colon, and on the sloughs; it has occasionally been detected in the mesenteric glands, which also swarm with micro-cocci. It has as yet not been found in the spleen, blood, or urine.

From the previous description, it will be seen that the resemblance of the bacillus of ulcerative colitis to the B. Coli and B. typhosus is in many respects very close. To bring

Tube I.

Tube II.

Tube III.

Shake Culture.

Shake Culture.

Surface Culture.

out this likeness and also the distinctive differences, my colleague has kindly tabulated for me the properties of the three bacilli which have just been engaging our attention. Most of the reactions he has verified himself, but one or two —as the formalin one—he has taken from the writings of others, with whose results also he has compared his own with regard to the B. Coli and B. typhosus.

Bacillus typhosus.	Bacillus Coli.	Bacillus of Ulcerative Colitis.
1. Colonies on gelatine are granular, with irregular edge, uniform thickness, of a greyish white colour, becoming greyish brown in time.	1. Colonies on gelatine are round or oval, with smooth, thin and transparent margins: often with concentric markings. Colonies are dirty white turning to yellowish brown.	1. Colonies on gelatine are spherical, thick, and glistening white in colour. Larger ones have thickened rim round the edge, which is regular.
2. Surface culture is translucent, greyish white, with irregular edge, and sharp margin, does not tend to spread much.	2. Dirty yellowish white growth, spreads considerably from point of inoculation.	2. Surface growth white. Does not tend to spread much; edge thickish; slightly indented, and sharply demarcated.
3. Stab culture, greyish white filament limited to track, becomes brownish film on surface, does not spread.	3. Light brown abundant growth along track, with branching arborescent tufts, gas bubbles developed, film spreads over the surface.	3. Growth along track presents coarse excrescences, white, raised, bead-like growth on surface.
4. Potato, moist and shiny, no visible growth.	4. Abundant soft shiny yellowish brown growth.	4. Well marked yellowish white growth.
5. Agar. Greyish white slimy layer, tends to spread.	5. Dirty yellowish white growth, tends to spread.	5. Well marked thick white growth; does not spread much.
6. Indol reaction negative.	6. Indol reaction present.	6. Indol reaction present.
7. Sterilized milk faintly acid, not coagulated, except after a long time.	7. Curdled in one to three days at body temperature.	7. Curdled in two to three days.
8. Bouillon, turbid in twenty-four hours at 28 C.	8. Bouillon, turbid in twenty-four hours at 38°C.	8. The same.
9. Gelatine not liquefied.	9. The same.	9. The same.

Bacillus typhosus.	Bacillus Coli.	Bacillus of Ulcerative Colitis.
10. Formalin (1 in 7000) in broth, no growth (Shild).	10. Formalin in broth, growth (Shild).	10.
11. Elsner, 1% Pot. iod. Potato gelatine, colonies small after twenty-four hours.	11. Colonies well developed in twenty-four hours.	11.
12. Litmus milk, no change in colour.	12. Pink colour developed, fading in time.	12. Pink colour developed, which spreads and is persistent.
13. Bacillus presents active movements.	13. Movements sluggish.	13. Movements sluggish or absent.
14. Decolourized by Gram's method.	14. The same.	14. Not decolourized by Gram's method, or only with difficulty.
15. Aërobic and facultative anaërobic.	15. The same.	15. Anaërobic propensities less pronounced.
16. Can be dried, and yields growth afterwards.	16. Cannot be dried.	16. Cannot be dried.
17. No gas in shake cultures.	17. Gas in shake culture.	17. Gas only observed occasionally.
18. Stains with difficulty.	18. Stains easily.	18. Takes on stain with avidity.
19. Agar treated with fuchsin is decolourized. (Gasser.)	19. The same. (Gasser.)	19.

It will be seen from the above that in many of its properties the B. of ulcerative colitis closely resembles those with which it has been compared. As regards its vitality, it is more powerful than either. Following the line of experiments quoted by Sternberg and Wathelet, in which the B. Coli and B. typhosus were grown together at the expense of the latter, Dr Goodliffe grew the B. Coli and the B. of ulcerative colitis together. Taking a growth in which the colonies of the two appeared to be fairly equal in number, he made shake cultures, and in a few generations the B. Coli had entirely disappeared.

Comparing now the B. of ulcerative colitis with organisms described by different writers as being the cause of various forms of dysentery, we find that Ogata's bacillus of Japan dysentery closely resembles it. One great point of distinction, however, is that the organism described by him liquefies gelatine, whereas the B. of ulcerative colitis does not. The B. of Chautemesse and Widal also resembles the B. of colitis; like it, it does not liquefy gelatine, but it stains badly. Possibly further investigation may show that these differences do not exist, and thus identify as the same, organisms which have hitherto been described as distinct.

Regarding the other organisms found in the stools of ulcerative colitis, mention may be made of the detection in one case of numerous large round or oval bodies with nuclei, which took on staining (Ehrlich-Weigert) to a most remarkable extent. They varied considerably in size, but were usually about three times the size of a red-blood corpuscle, and possessed from one to four nuclei. The body of the cell stained only faintly. Dr Hamilton describes curious bodies of a similar nature occurring in a case of English dysentery.

The B. Coli was frequently present, and various bacilli were met with from time to time closely resembling it, but differing in some mode of growth, reaction, etc. In addition, many forms of micro-cocci were found. The pyogenes aureus was present in several cases, and also other gelatine liquefying micro-cocci. A bacillus giving all the characters of the B. proteus vulgaris was also frequently present. Importance is to be attached to the presence of these swarms of micro-organisms, for it is possible that they have a greater bearing on the question of causation than is generally supposed. Klein and others have pointed out that there is often a "mixed infection" in enteric fever, and have advanced the theory that the streptococci and other septic organisms so often present prepare the mucous membrane of the intestine for the specific microbe. May the same view not hold in the case of ulcerative colitis? It has already been pointed out that it

attacks chiefly the old and feeble, and those whose vitality is lowered by arterial degeneration, etc. Moreover, we have often observed a remarkable tendency to the development of erysipelas and cellulitis in feeble general paralytics and epileptics after some slight cut or abrasion. In this class of cases there is a greatly lowered vitality, and septic organisms increase and develop whenever the slightest hold is given them. I mention these facts not with the view of drawing any definite conclusions, but merely as showing the tendency there is for disease dependent upon micro-parasites to attack the feeble insane when associated together in large numbers. Regarding the staphylococcus pyogenes aureus, which was found in great numbers in six cases, Netter has observed its presence in fæces, but Bierstock says that micro-cocci of all kinds are absent or scanty in the healthy bowel.

Sternberg, in his recent work on bacteriology, advances a most interesting theory to account for certain cases of dysentery.

He says, besides the presence of undoubted infection in primary dysentery, there is also probably an acquired one developed from primary diarrhœa, just as there is a probable occurrence of progressive development in the property of infectiveness in simple sore throat up to a condition of diphtheria. In concluding these remarks upon the bacteriological investigation of this ulcerative colitis, I may remark that it is a source of great regret that Dr Goodliffe has been unable to verify the specificity of the bacillus by the crucial test of reproduction of the same disease by inoculation.

Before leaving the subject of the ætiology of dysentery, I should like briefly to allude to the question of fæcal retention, and what, if any, influence it possesses as a causative factor. Many writers of note attach great, and I think undue, importance to constipation and fæcal accumulations in this connection. Ziegler says a colitis may be caused by it, which, however, is not specific. Strümpell says, persistent fæcal impaction of the rectum by a purely mechanical effect may

excite a diphtheritic inflammation similar to the anatomical changes in dysentery. In Fagge's system of medicine, Virchow is quoted as attributing to stagnation of fæces in the cæcum and flexures, the peculiar liability of these parts to be attacked ; and Annesley, in the same work, says that in India the disease often commences with the characteristic signs of morbid accumulation in the colon. Trousseau also attaches importance to the presence of scybalous masses. The idea uppermost in the minds of these writers appears to be, that the fæces by accumulation produce a direct mechanical effect, intensified no doubt by the greater power of producing irritation which prolonged retention gives them. The suggestion is a natural one, and no doubt many instances might be adduced of serious injury inflicted on the wall of the gut by fæcal accumulation. In such cases, however, the damage done is strictly confined to a definite and usually limited portion of the bowel, most frequently the sigmoid. The nature, moreover, of the case is usually revealed by the history, and a physical examination of the abdomen will, where the accumulation is sufficient, detect the presence of a fæcal tumour. Where, however, there is no such tumour, but simply a history of prolonged and obstinate constipation, the diagnosis is usually sufficiently easy, and in no instance at any rate do we meet with that assemblage of symptoms which characterises dysentery proper. A more frequent change associated with chronic constipation than this localized colitis is dilatation and thinning of the walls of the colon, and another less common affection has been described as membranous colitis. Numerous other objections may be urged which tend to throw doubt on the view which accepts fæcal retention as a cause of dysentery. Constipation is such a common condition, and is, moreover, so incapable of explaining the presence or absence of endemic dysentery or the limited epidemics in institutions and special localities in towns, that its inclusion nowadays as a cause of dysentery must be owing to the authority and weight attached to the

name of its advocates. Constipation and feculent accumulation are as frequent now as formerly, and yet, why is dysentery, comparatively speaking, in this country so rare? Speaking as to the ætiological importance of this condition in dysentery in tropical climates, so far as my own limited experience goes, and that accorded me by others, constipation, from the altered mode of living and the excessive perspiration from the heat, appears very common among the Europeans, and to attach anything but an accidental value to its presence in dysentery where that disease is endemic, and is probably caused by insanitary conditions or contaminated drinking water, is, to say the least of it, selecting a most unlikely cause. That it does induce a local colitis, and even occasionally one accompanied by a membranous exudation, is not disputed, but such must not be confounded with a disease the cause or causes of which investigation tends to show are so utterly different. To indicate that the contents of the intestine have any influence other than a mechanical one in inducing a colitis, and that non-specific, implies a belief which I think is not warranted. The mucous lining, from the extreme variety of the ingesta, must be accustomed to innumerable chemical substances, many of them identical with those supposed to cause the inflammatory conditions to be met with in feculent accumulation. On the other hand, we know that ptomaines which are formed by the action of micro-organisms on proteids, and must invariably be present in the intestinal contents, and which in addition are often introduced along with food, are capable under certain circumstances of inducing poisoning with symptoms of violent gastro-intestinal irritation and inflammation. It is offered as explanatory of the escape from this daily risk of poisoning that normally these substances are not absorbed by the intestines, but escape in the fæces. That absorption, however, may and does take place is evidenced by their detection in the urine. Now the question presented is, under what circumstances does absorption of these substances take place? Unfortunately this is more

easily propounded than answered. Why putrid meat or fish may one time be consumed with impunity and at another cause symptoms of poisoning with gastro-intestinal inflammation is difficult to explain I would suggest, however, in the cases in which the poison has entered with the ingesta, that, owing to a special intensity it has acted as a direct irritant, and that absorption is only permitted when the mucous coat is eroded or otherwise altered by the inflammation set up. In this way also retained fæces by their mechanical effect may cause laceration of the lining membrane, and thus permit of local inflammation being set up by the micro-organisms present with septic absorption; that, in fact, it is a septic inoculation as occurs in traumatic erysipelas, phlegmonous cellulitis, etc.

It may perhaps not be inappropriate to discuss in this chapter the theory which assigns a trophic origin to ulcerative colitis in asylums. I purposely confine my remarks to cases occurring in these institutions, as it is there the disease is most frequently met with, and the instances elsewhere recorded in which this ulcerated condition of the large bowel is associated with cerebral or spinal disease are too few to warrant any deduction being drawn. In the *Journal of Mental Science*, April, 1895, Dr Cowan advanced this theory, somewhat ruthlessly criticised by Dr Eurich, the pathologist at Whittingham Asylum; and Dr Hale White (Guy's Hosp. Rep.) relates one and quotes two cases of ulcerative colitis recorded by Dr Acland, in which a trophic origin was noted. There are, however, in my opinion, insuperable objections to this view, and to these I propose to direct attention.

Dr Cowan indicates that general paralytics are specially liable to be attacked. So far as I am aware, he is the first to make this assertion—though Dr Clouston, in his account of the epidemic of dysentery in the Cumberland and Westmoreland Asylum, mentions the fact that three out of eight general paralytics were attacked—but it is certainly not our experience here. A reference to Table I. shows that out of

nearly 500 deaths from general paralysis, only thirteen died of ulcerative colitis. We see, therefore, that the number dying of this colitis, even where the mortality among that class of the insane accounts for as much as even a third of the total deaths in a year, is comparatively small. Again, taking the number of deaths (amounting perhaps to 200 or more) among the same class over an extended period of years in other asylums, one will often find that there is not a solitary instance figuring in the death tables of death from dysentery, or—accepting the view of those who regard the bowel affection as different from dysentery—from simple ulcerative colitis. How are we to explain by this theory the marked absence of trophic colitis in some asylums and its almost endemic prevalence in others? Trophic processes are no respecters of persons, and do not confine themselves to one asylum more than another. We would, therefore, not unnaturally expect to find with more or less frequency this trophic intestinal lesion in every asylum in which general paralytics were at all numerous. Amongst this class, as compared with other types of insanity, trophic lesions, I admit, are by far the most frequently met with, and differ little in their mode of onset, site and progress from those occurring in the sane as a result of myelitis, cerebral softening and hæmorrhage. They are almost invariably preceded by epileptiform or congestive attacks, with high temperature often within an hour of the seizure. The temperature only has claims to be admitted as a symptom of the bowel affection, and one, moreover, which is not always present in the latter, otherwise I can see no connection between the colitis and trophic manifestations in other regions of the body. Nor am I aware that the onset of the bowel affection is heralded by convulsive or congestive phenomena. We would also expect, at least occasionally, if the colitis were trophic, to find it associated with similar lesions elsewhere—in the kidneys and bladder, and in the sacral and gluteal regions—such as are met with in other affections

of the central nervous system, and to which allusion has already been made. Of the many trophic lesions I have witnessed in the insane, I can recall comparatively few indeed in which they were associated with ulcerative colitis, and from the clinical history and other circumstances, I considered them to be in no way connected. These facts, then, seem to militate against the view that ulcerative colitis in the insane is a trophic lesion, and is to be found for the most part in general paralytics.

CHAPTER VI

In bringing to a close the remarks upon dysentery in asylums, it will perhaps be of advantage to recapitulate briefly the chief features of the disease, and also to discuss with a little more detail some points in connection with it to which importance is to be attached, and which have already been but slightly dwelt upon. It will also give me an opportunity of explaining, perhaps more clearly than I have, my reasons for stating that this colitis as it appears in asylums is not, as is often stated, a simple colitis, but is nothing more nor less than dysentery. Following the classification adopted in the text, and using the term ulcerative colitis as synonymous with dysentery, I have called the affection idiopathic ulcerative colitis, as distinguishing it from the secondary dysenteric ulcerations or colitis occurring in the course of other diseases and various poisonings. Without repeating the definition which has already been given, we may state briefly that it is an acute febrile disease, attacking chiefly the feeble and debilitated, and those past the prime of life, usually terminating in about a fortnight, and characterised during some part of its course, or even from the outset, by scanty and frequent stools of mixed mucus and blood, which, towards a fatal termination, often become puriform. It is, as a perusal of asylum reports will show, often associated with overcrowding, and is sometimes endemic.

We also find that when present, erysipelas, phlegmonous

erysipelas, tardy and suppurative healing of wounds, sometimes ending in septic and pyæmic conditions, notwithstanding every precaution—all of common enough occurrence in the early history of our hospitals in large towns, and all pointing to an undesirable state of the atmosphere and an unfavourable class of patients—are often to be met with. The presence of these infective diseases and the unhappy healing of wounds in great measure depend on the physique of the patients, and where there are many feeble paralytics, epileptics and dements, to those acquainted with the habits of these types of insanity, the appearance of the diseases and conditions alluded to does not, however, occasion the surprise it otherwise would.

As elsewhere indicated, many asylums are only occasionally visited by outbreaks of dysentery and erysipelas, and we have evidence that when such occur in asylums hitherto or for a long time exempt, a definite cause is usually discovered, one known to give rise to these diseases — contaminated drinking water, air contaminated by sewage, etc.—and with whose removal it disappears. Instances of this may be adduced in such outbreaks as happened in the Millbank Penitentiary and in the Cumberland and Westmoreland Asylum in 1864. And a reference to the account of dysentery in Dublin towards the latter end of 1818 convinces me that in its essentials it is a fairly accurate description of a disease which at the present time is encountered in some asylums, and which was then known and described as dysentery. For reasons already specified, dysentery, unless imported from abroad, is now in this country comparatively seldom met with, but the same causes which in times past rendered outbreaks common enough occurrences, still exist and still occasion its appearance. In the last report of the Commissioners in Lunacy, they state that colitis appeared in seventeen asylums, and dysenteric diarrhœa in ten, and that in some instances an epidemic character was assumed. The fact that reports of

institutions for the insane and the annual blue-book compiled by the Commissioners in Lunacy are seldom or never read by medical men other than those directly interested in the care and treatment of lunatics, may in great measure account for their lack of information as to the more frequent occurrence of dysentery in this country than is generally suspected. Owing, however, to the increased attention which of recent years has been directed towards the treatment of the insane and to the care exercised in the construction of new and the improvement of old asylums, knowledge on this point is becoming more general. We know the influence which unwholesome food exerts in the production of scurvy and purpura, and the fact that they and dysentery have in times past often been associated together, and often complicated each other, should direct our attention in this direction as a possible contributory cause of the colitis. That a close connection exists between the two former is undoubted, and from their frequent association with dysentery, a possible relationship between the three diseases is conceivable, closer, at any rate, than is at present recognised. Fortunately, scorbutic dysentery is never seen, but I am not so positive that certain diets may not induce a state of malnutrition which, with the presence of causes calculated to induce dysentery, along with the general feebleness of many of the patients, gives the disease that asthenic character so peculiar to many of the cases.

In conclusion, I venture to affirm that, apart from its specific cause, which we have strong evidence to show is dependent upon a micro-organism—a bacillus, this ulcerative affection of the colon is clinically and anatomically dysentery.

THE END

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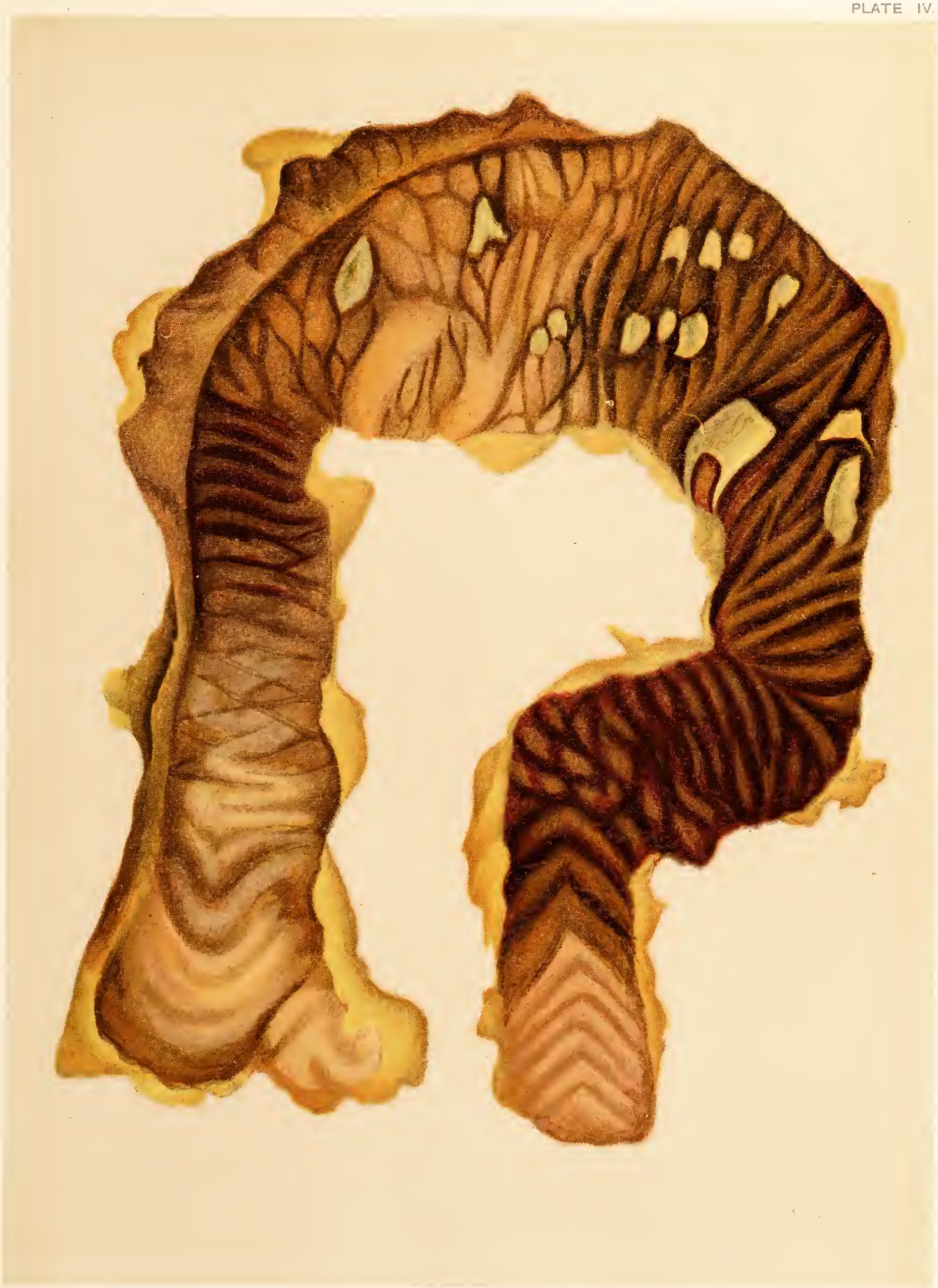
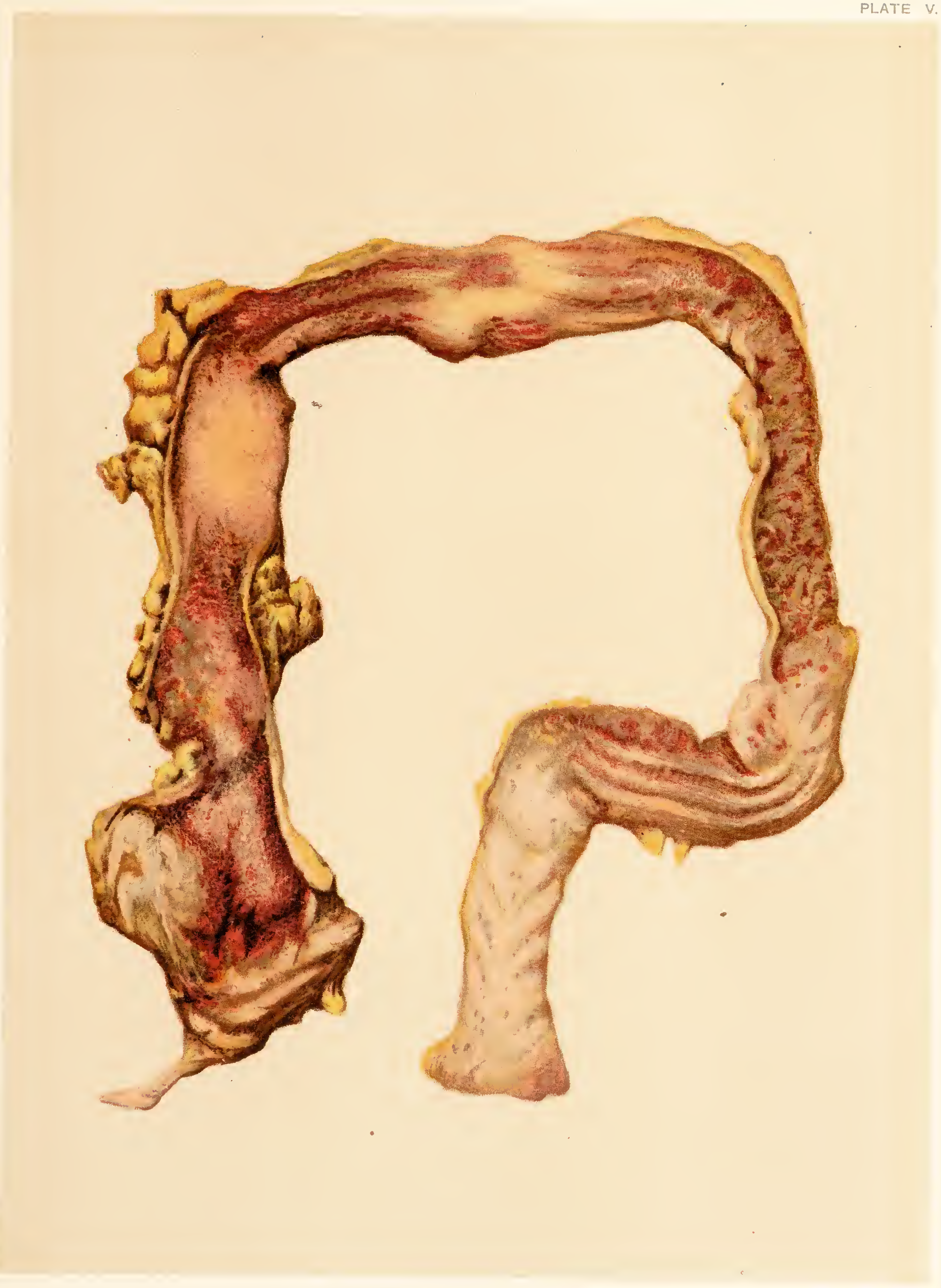
Acute Asthenic Dysentery of 11 days duration.

Rectum from a case of Diphtheritic Dysentery of 14 days duration.

Acute Sthenic Dysentery of 14 days duration.



Acute Sthenic Dysentery of 12 days duration.



Chronic Dysentery of over a year's duration.